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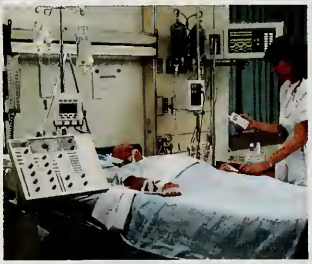

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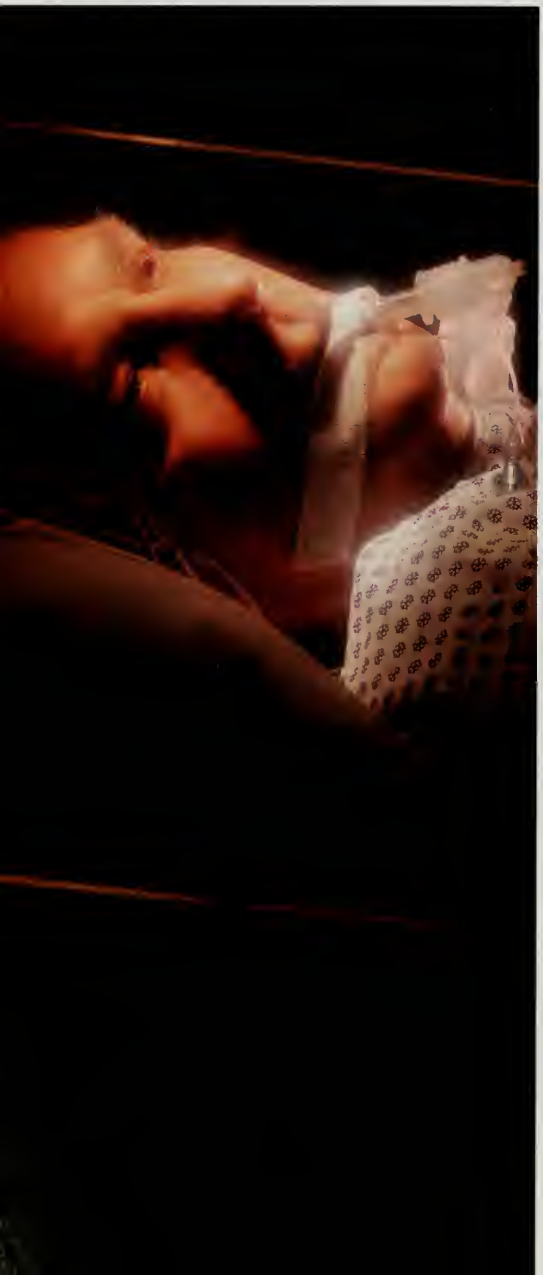

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Trauma Systems: A Model for Regionalized Care (editorial)—D Trunkey. JAMA 1995;273(5):421-422. (Pertains to the paper by Bazzoli et al abstracted on Page 208.)

The Pulmonary Complications of Crack Cocaine: A Comprehensive Review—DY Haim, ML Lippmann, SK Goldberg, MD Walkenstein. Chest 1995;107:233-240.

Acute Laryngeal Obstruction (review)—DA Ross, CT Sasaki. Lancet 1994;344 Dec 24/31:1743-1748.

Cardiopulmonary Resuscitation and Older Adults' Expectations—MD Godkin, EL Toth. Gerontologist 1994;34(6):797-802.

Chest Physiotherapy and Cystic Fibrosis: Why is the Most Effective Form of Treatment Still Unclear? (review)—MT Williams. Chest 1994;106:1872-1882.

A Proposal for Structured Reporting of Randomized Controlled Trials (special communication)—Standards of Reporting Trials Group. JAMA 1994;272(24):1926.

Health Care Reform and Older Adults (symposium proceedings)—GD Cohen. Gerontologist 1994;34(5):584-619.

A Comparison of Four Methods of Weaning Patients from Mechanical Ventilation—A Esteban, F Frutos, MJ Tobin, I Alía, JF Solsona, I Valverdú, et al. N Engl J Med 1995;332:345.

BACKGROUND: Weaning patients from mechanical ventilation is an important problem in intensive care units. Weaning is usually conducted in an empirical manner, and a standardized approach has not been developed. METHODS: We carried out a prospective, randomized, multicenter study involving 546 patients who had received mechanical ventilation for a mean (±SD) of 7.5 ± 6.1 days and who were considered by their physicians to be ready for weaning. One hundred thirty patients had respiratory distress during a two-hour trial of spontaneous breathing. These patients were randomly assigned to undergo one of four weaning techniques: intermittent mandatory ventilation, in which the ventilator rate was initially set at a mean (±SD) of 10.0 ± 2.2 breaths per minute and then decreased, if possible, at least twice a day, usually by 2 to 4 breaths per minute (29 patients); pressure-support ventilation, in which pressure support was initially set at 18.0 ± 6.1 cm of water and then reduced, if possible, by 2 to 4 cm of water at least twice a day (37 patients); intermittent trials of spontaneous breathing, conducted two or more times a day if possible (33 patients); or a once-daily trial of spontaneous breathing (31 patients). Standardized protocols were followed for each technique. RESULTS: The median duration of weaning was 5 days for intermittent mandatory ventilation (first quartile, 3 days; third quartile, 11 days), 4 days for pressure-support ventilation (2 and 12 days, respectively), 3 days for intermittent (multiple) trials of spontaneous breathing (2 and 6 days, respectively), and 3 days for a once-daily trial of spontaneous breathing (1 and 6 days, respectively). After adjustment for other covariates, the rate of successful weaning was higher with a once-daily trial of spontaneous breathing than with intermittent mandatory ventilation (rate ratio, 2.83; 95 percent confidence interval, 1.36 to 5.89; $P < 0.006$) or pressure-support ventilation (rate ratio, 2.05; 95 percent confidence interval, 1.04 to 4.04; $P < 0.04$). There was no significant difference in the rate of success between once-daily trials and multiple trials of spontaneous breathing. CONCLUSIONS: A once-daily trial of spontaneous breathing led to extubation about three times more quickly than intermittent mandatory ventilation and about twice as quickly as pressure-support ventilation. Multiple daily trials of spontaneous breathing were equally successful.

An Expanded Scoring System Including an Index of Nutritional Status for Patients with Cystic Fibrosis—ME Cabrera, MD Lough, CF Doershuk, AE Salvator. Pediatr Pulmonol. 1994;18:199.

Evaluation of success or failure of therapy for patients with cystic fibrosis (CF) commonly relies on the results of a single pulmonary function test (PFT). Most PFT measurements reflect different functional aspects of the lung. Although no single parameter can summarize all aspects of lung function, a combination of several may provide an advantage by reflecting

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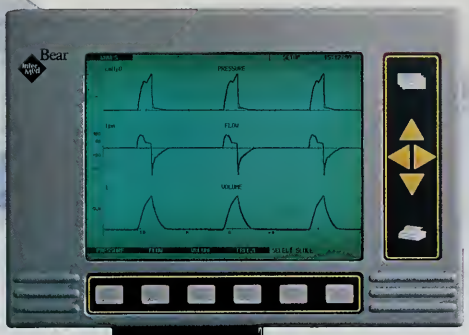

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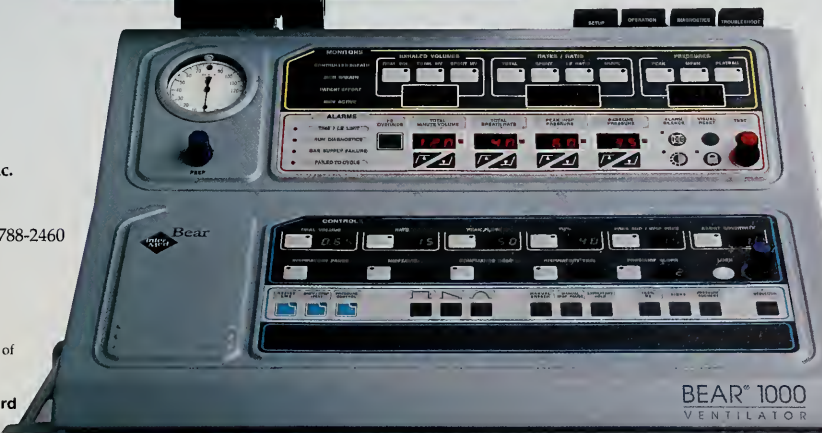

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the overall abnormality of lung function in one number. Cropp et al. (1982, Am Rev Respir Dis 126:211-216) developed a multiparameter pulmonary function score (PFS) using the results of six separate parameters obtained from a PFT. In CF, there is also the potential for declining nutritional status (NS), leading to malnutrition and skeletal muscle wasting. Our aim was to expand the PFS by including weighted information on NS, and to determine whether the expanded score (nutritional-pulmonary function score, NPFS) was more sensitive in detecting change in outcome variables than the PFS. Individual PFT parameters, percent ideal body weight (%IBW), and an index of anaerobic performance (AP) were measured in 21 patients on admission to the hospital and again at discharge. In the group as a whole, in-hospital therapy resulted in improvement ($P < 0.01$) in individual PFT parameters, %IBW, PFS, and NPFS, and no change in AP. While the PFS more effectively reflected improvement in lung function than did any single PFT parameter, the NPFS resulted in an even more sensitive index of change. Based on these results, we believe that the NPFS, which includes both lung and nutritional status, provides an effective and sensitive index of disease severity that can be used as a unifying measure to: 1) detect disease progression; 2) guide rehabilitation and training; 3) stratify patients for clinical trials; or 4) evaluate the effects of a therapeutic intervention.

Physical Activity and Public Health: A Recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine—RR Pate, M Pratt, SN Blair, WL Haskell, CA Macera, C Bouchard, et al. JAMA 1995; 273:402.

OBJECTIVE: To encourage increased participation in physical activity among Americans of all ages by issuing a public health recommendation on the types and amounts of physical activity needed for health promotion and disease prevention. PARTICIPANTS: A planning committee of five scientists was established by the Centers for Disease Control and Prevention and the American College of Sports Medicine to organize a workshop. This committee selected 15 other workshop discussants on the basis of their research expertise in issues related to the health implications of physical activity. Several relevant professional or scientific organizations and federal agencies also were represented. EVIDENCE: The panel of experts reviewed the pertinent physiological, epidemiologic, and clinical evidence, including primary research articles and recent review articles. CONSENSUS PROCESS: Major issues related to physical activity and health were outlined, and selected members of the expert panel drafted sections of the paper from this outline. A draft manuscript was prepared by the planning committee and circulated to the full panel in advance of the 2-day workshop. During the workshop, each section of the manuscript was reviewed by the expert panel. Primary attention was given to achieving group consensus concerning the recommended types and amounts of physical activity. A concise "public health message" was developed to express the recommendations of the panel. During the ensuing months, the consensus statement was further reviewed and revised and was formally endorsed by both the Centers for Disease Control and Prevention and the American College of Sports Medicine. CONCLUSION: Every US adult should accumulate 30 minutes or more of moderate-intensity physical activity on most, preferably all, days of the week.

Progress in the Development of Trauma Systems in the United States: Results of a National Survey—GJ Bazzoli, KJ Madura, GF Cooper, EJ MacKenzie, RV Maier. JAMA 1995;273:295.

OBJECTIVE: To examine the status of trauma system development and key structural and operational characteristics of these systems. DESIGN AND SETTING: National survey of trauma systems with enabling state statute, regulation, or executive orders and for which designated trauma centers were present. PARTICIPANTS: Trauma system administrators and directors of 37 state and regional organizations that had legal authority to administer trauma systems, which represented a response rate of 90.2%. MAIN OUTCOME MEASURES: Trauma system components that had been implemented or were under development. RESULTS: From 1988 to 1993, the number of states meeting one set of criteria for a complete trauma system criteria increased from two to five. The most common deficiency in establishing trauma systems was failure to limit the number of designated trauma centers based on community need. Although most existing trauma systems have developed formal processes for designating trauma centers, prehospital triage protocols to allow hospital bypass, and centralized trauma registries, several systems lack standardized policies for interhospital transfer and systemwide evaluation. CONCLUSION: State and regional organizations have accomplished a great deal but still have substantial work ahead in developing comprehensive trauma systems. Research is needed to better understand the relationship between trauma volume and outcomes of care as well as the impact of trauma system structure and operational characteristics on care delivery. Improved measures of patient outcome are also needed so that effective system evaluation can take place.

Effect of Corticosteroids for Fetal Maturation on Perinatal Outcomes—NIH Consensus Development Panel on the Effect of Corticosteroids for Fetal Maturation on Perinatal Outcomes. JAMA. 1995;273:413.

OBJECTIVE: To develop a consensus on the use of antenatal corticosteroids for fetal maturation in preterm infants. PARTICIPANTS: A nonfederal, nonadvocate, 16-member consensus panel including representatives from neonatology, obstetrics, family medicine, behavioral medicine, psychology, biostatistics, and the public; 19 experts in neonatology, obstetrics, and pharmacology presented data to the consensus panel and a conference audience of approximately 500. EVIDENCE: An extensive bibliography of references was produced for the consensus panel and the conference audience using a variety of on-line databases including MEDLINE. The consensus panel met several times prior to the conference to review the literature. It also commissioned an updated meta-analysis, a neonatal registry review, and an economic analysis that were presented at the conference. The experts prepared abstracts for distribution at the conference, presented data, and answered questions from the panel and audience. The panel evaluated the strength of the scientific evidence using the grading system developed by the Canadian Task Force on the Periodic Health Examination and adapted by the US Preventive Services Task Force. CONSENSUS: The consensus panel, answering predefined consensus questions, developed their conclusions based on the scientific evidence presented in open forum and the scientific literature. CONSENSUS STATEMENT: The consensus panel composed a draft statement that was read in its entirety at the conference for comment. The panel released a revised statement at the end of the conference and finalized the revisions a few weeks after the conference. CONCLUSIONS: Antenatal corticosteroid therapy is indicated for women at risk of premature delivery with few exceptions and will result in a substantial decrease in neonatal morbidity and mortality, as well as substantial savings in health care costs. The use of antenatal corticosteroids for fetal maturation is a rare example of a technology that yields substantial cost savings in addition to improving health.

Empirical Evidence of Bias: Dimensions of Methodological Quality Associated with Estimates of Treatment Effects in Controlled Trials—KF Schulz, I Chalmers, RJ Hayes, DG Altman. JAMA, 1995;273:408.

OBJECTIVE: To determine if inadequate approaches to randomized controlled trial design and execution are associated with evidence of bias in estimating treatment effects. DESIGN:

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REFERENCES 1 Data on file HealthScan Products Inc. 2 Wheeler BB, Bollers ND: Initial experience with a novel spacer device (Presented at the 1993 meeting of the American Academy of Allergy and Immunology) * U.S. Patent No. 5,040,527 OA760004-0 © 1994, HealthScan Products Inc

An observational study in which we assessed the methodological quality of 250 controlled trials from 33 meta-analyses and then analyzed, using multiple logistic regression models, the associations between those assessments and estimated treatment effects. DATA SOURCES: Meta-analyses from the Cochrane Pregnancy and Childbirth Database. MAIN OUTCOME MEASURES: The associations between estimates of treatment effects and inadequate allocation concealment, exclusions after randomization, and lack of double-blinding. RESULTS: Compared with trials in which authors reported adequately concealed treatment allocation, trials in which concealment was either inadequate or unclear (did not report or incompletely reported a concealment approach) yielded larger estimates of treatment effects ($P < 0.001$). Odds ratios were exaggerated by 41% for inadequately concealed trials and by 30% for unclearly concealed trials (adjusted for other aspects of quality). Trials in which participants had been excluded after randomization did not yield larger estimates of effects, but that lack of association may be due to incomplete reporting. Trials that were not double blind also yielded larger estimates of effects ($P = 0.01$), with odds ratios being exaggerated by 17%. CONCLUSIONS: This study provides empirical evidence that inadequate methodological approaches in controlled trials, particularly those representing poor allocation concealment, are associated with bias. Readers of trial reports should be wary of these pitfalls, and investigators must improve their design, execution, and reporting of trials.

Global Epidemiology of Tuberculosis: Morbidity and Mortality of a Worldwide Epidemic—MC Raviglione. DE Snider. Jr. A Kochi. JAMA. 1995;273:220.

This article describes the global epidemiology of tuberculosis and reviews recent estimates of tuberculosis incidence and mortality in the world. The highest prevalence of tuberculosis infection and estimated annual risk of tuberculosis infection are in sub-Saharan Africa and Southeast Asia. Overall, almost 3.8 million cases of tuberculosis were reported in the world in 1990, of which 49% were in Southeast Asia. From the period 1984 through 1986 to the period 1989 through 1991, notification rates increased in all World Health Organization regions, except the American and the European regions. In 1990, there were an estimated 7.5 million cases of tuberculosis and 2.5 million deaths worldwide. The human immunodeficiency virus epidemic is causing increases in the number of tuberculosis cases, particularly in Africa, although increases are also expected in Southeast Asia. In many industrialized countries, tuberculosis has recently failed to decline and in eastern Europe and the former Soviet Union, cases and deaths are increasing. Drug resistance is a serious problem, especially in the United States. If worldwide control of tuberculosis does not improve, 90 million new cases and 30 million deaths are expected in the decade 1990 through 1999.

A Critical Appraisal of the Quality of Quality-of-Life Measurements—TM Gill, AR Feinstein. JAMA 1994;272:619.

OBJECTIVE: To evaluate how well quality of life is being measured in the medical literature and to offer a new approach to the measurement. DATA SOURCES: Original English-language articles having the term "quality of life" in their titles were identified from a recent Quality-of-Life Bibliography and from two MEDLINE searches. Articles were eligible for review only if they described or used one or more "quality-of-life" instruments. STUDY SELECTION: Twenty-five articles were randomly selected from each of the three data sources. DATA EXTRACTION: Each article was reviewed for its compliance with two sets of criteria having several components, which are cited under "Data Synthesis." DATA SYNTHESIS: (1) Investigators conceptually defined quality of life in only 11 (15%) of the 75 articles; identified the targeted domains in only 35 (47%); gave reasons for selecting the chosen quality-of-life instruments in only 27 (36%); and aggregated their results into a composite quality-of-life score in only 27 (38%) of 71 eligible articles. (2) No article distinguished "overall" quality of life from health-related quality of life; patients were invited to give their own separate rating for quality of life in only 13 articles (17%); and among 71 eligible articles, patients were asked to supplement the stipulated items with personal responses in only nine (13%) and to rate the importance of individual items in only six (8.5%). CONCLUSIONS: Because quality of life is a uniquely personal perception, denoting the way that individual patients feel about their health status and/or nonmedical aspects of their lives, most measurements of quality of life in the medical literature seem to aim at the wrong target. Quality of life can be suitably measured only by determining the opinions of patients and by supplementing (or replacing) the instruments developed by "experts."

Cardiopulmonary Resuscitation and Rib Fractures in Infants: A Postmortem Radiologic-Pathologic Study—MR Spevak, PK Kleinman, PL Belanger, C Primack, JM Richmond. JAMA. 1994;272:617.

OBJECTIVE: To determine the incidence of rib fractures visible at autopsy or with postmortem radiographs after cardiopulmonary resuscitation (CPR) in infants younger than 1 year. DESIGN: Retrospective review. SETTING: Medical examiner's office in a county consisting of a medium-sized city, towns, and rural areas. PATIENTS: Ninety-one infants (56 males, 35 females; mean age, 2.4 months; age range, 26 hours to 8.5 months) without evidence of child abuse who had undergone CPR before death. METHODS: Medical records, skeletal surveys, and autopsy results were reviewed. RESULTS: No patient had rib fractures. CONCLUSIONS: Cardiopulmonary resuscitation is unlikely to cause rib fractures in infants.

Critical Respiratory Events in the Post-Anesthesia Care Unit: Patient, Surgical, and Anesthetic Factors—DK Rose, MM Cohen, DF Wigglesworth, DP DeBoer. Anesthesiology 1994;81:410.

BACKGROUND: Previous studies have noted a high incidence of adverse outcomes in the postanesthesia care unit (PACU), but few have examined associated factors and patient outcomes. To determine the frequency of acute, unanticipated respiratory problems and to examine the associated patient, surgical, and anesthetic factors, we prospectively collected preoperative, intraoperative, and postoperative data on 24,157 consecutive PACU patients who received a general anesthetic during a 33-month period. METHODS: A PACU critical respiratory event (CRE) was defined as any unanticipated hypoxemia (hemoglobin oxygen saturation < 90%), hypoventilation (respiratory rate < 8 breaths/min or arterial carbon dioxide tension > 50 mm Hg) or upper-airway obstruction (stridor or laryngospasm) requiring an active and specific intervention (ventilation, tracheal intubation, opioid or muscle relaxant antagonism, insertion of oral/nasal airway or airway manipulation). These problems were documented by PACU nurses whereas data on case-mix, surgical factors, and intraoperative management were retrieved from the anesthetic record. Significant patient, surgical, and anesthetic factors were identified by logistic regression analysis. Other morbidity experienced by patients with a CRE was also noted. RESULTS: For patients given general anesthesia the risk of a CRE was 1.3% (hypoxemia 0.9%, hypoventilation 0.2%, airway obstruction 0.2%). Preoperative factors that increase risk were age > 60 yr, male gender, diabetes, and obesity ($P < 0.05$). Patients who underwent operative procedures on an emergency basis and whose operation was longer than 4 h were also at increased risk, but those undergoing perineal procedures were at lower risk ($P < 0.05$). Anesthetic risk factors ($P < 0.05$) included opioid premedication (relative odds 1.8), sedatives preoperatively (2.0), fentanyl > 2.0 $\mu g \cdot kg^{-1} \cdot h^{-1}$ as the sole opioid (1.9), fentanyl used in combi-

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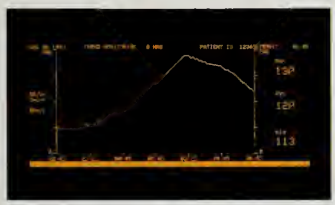

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nation with morphine (1.6) and atracurium ≥ 0.25 mg · kg^{-1} · h^{-1} (2.2). Patients in whom anesthesia was induced with thiopental (relative odds 2.5), compared with those who received propofol for induction, were also at increased risk of a CRE. Patients with a CRE stayed longer in PACU, had higher rates of unanticipated admissions to the intensive care unit, and were more likely to have PACU cardiac problems ($P < 0.01$). CONCLUSIONS: A CRE is relatively rare. Multiple patient and surgical factors and specific aspects of anesthetic management are associated with the occurrence of a CRE in the PACU.

Automatic Weaning from Mechanical Ventilation Using an Adaptive Lung Ventilation Controller—DM Linton, M Phil, PD Potgieter, S Davis, ATJ Fourie, JX Brunner, TP Laubscher. Chest 1994;106:1843.

STUDY OBJECTIVE: To evaluate a new method of closed-loop mechanical ventilation using an adaptive lung ventilation (ALV) controller in patients with different pathologic causes of respiratory failure at a time when they first met standard weaning criteria. STUDY DESIGN: Prospective, open, selected case study. SETTING: The 10-bed, multidisciplinary respiratory intensive care unit at Groote Schuur hospital, which is a teaching unit of the University of Cape Town. PATIENTS: Twenty-seven patients (9 patients in each of 3 groups: normal lungs, parenchymal lung disease, and COPD) who required prolonged mechanical ventilation and who met standard weaning criteria were included. Our institutional committee for ethical research approved the study and informed consent was obtained. INTERVENTIONS: The patients were mechanically ventilated and had daily measurements of vital capacity, respiratory rate, and arterial blood gas analysis until they met standard weaning criteria. On the day that each patient met the weaning criteria, a closed-loop control algorithm providing ALV was implemented on a modified ventilator (Hamilton AMADEUS) with a PC-based lung function analyzer. After measuring gross alveolar ventilation, patients were placed in ALV and ventilatory and hemodynamic parameters were measured at baseline, 5 min, 30 min, and 2 h. Pertinent parameters measured included airway pressures, pressure support levels, respiratory rates, rapid shallow breathing indices, airway resistance indices, and patient respiratory drive and work indices. MEASUREMENTS AND RESULTS: In 22 patients, ALV reduced pressure support to 5 cm H_2O and an intermittent mandatory ventilation rate of 4 breaths/min within 30 min, and all but 1 of these patients were successfully extubated within 24 h. In four patients, pressure support was maintained by ALV at a mean level of 14.6 cm $H_2O \pm$ for 2 h and these patients were recorded as having failed to wean. There was a measurable difference in an index of airway resistance relative to muscular activity between the successfully weaned and failed wean patients with COPD during the attempted wean by the ALV controller. CONCLUSIONS: ALV will provide a safe, efficient wean and will respond immediately to inadequate ventilation in patients when standard weaning criteria are met.

Transmissibility of *Pseudomonas cepacia* Infection in Clinic Patients and Lung-Transplant Recipients with Cystic Fibrosis—S Steinbach, L Sun, R-Z Jiange, P Flume, P Gilligan, TM Egan, R Goldstein. N Engl J Med 1994;331:981.

BACKGROUND: In patients with cystic fibrosis, infection with *Pseudomonas cepacia* is associated with poor outcomes. However, the extent of person-to-person transmission and the source of *P cepacia* infection after lung transplantation are not well defined. Using DNA-based typing systems, we sought to determine the genetic relatedness of *P cepacia* infection at one cystic fibrosis center. METHODS: We analyzed 65 *P cepacia* isolates gathered over a period of eight years at a single cystic fibrosis center from 17 clinic patients and from 5 patients who underwent double-lung transplantation. The isolates were analyzed by ribotyping and chromosomal fingerprinting based on pulsed-field gel electrophoresis. RESULTS: Analyses of serial isolates revealed that each clinic patient and transplant recipient harbored a different *P cepacia* clone that was persistent. In the transplant recipients, the preoperative and postoperative isolates were identical. In the two patients with disseminated infection after lung transplantation, isolates from multiple sites were identical and indicated clonal expansion of the previous respiratory *P cepacia* strain. Pulsed-field gel electrophoresis proved both more discriminative and more practical than ribotyping as a means of defining the genetic relatedness of the *P cepacia* isolates. CONCLUSIONS: Our serial analyses in patients with cystic fibrosis at one center found distinct strains of *P cepacia* persistently infecting each patient and no evidence of person-to-person transmission of this organism. *P cepacia* infection after lung transplantation was due to the persistence of the strain present before transplantation.

Sleep and Nocturnal Mouthpiece IPPV Efficiency in Postpoliomyelitis Ventilator Users—JR Bach, AS Alba. Chest 1994; 106:1705.

STUDY OBJECTIVE: Intermittent positive pressure ventilation (IPPV) can be delivered via various oral, nasal, or oronasal interfaces as an alternative to tracheostomy for up to 24 h of ventilatory support. Nocturnal nasal IPPV is often associated with frequent transient but at times severe oxyhemoglobin desaturations ($dS_{aO_2}s$) and sleep fragmentation. The purpose of this study was to determine if nocturnal mouthpiece IPPV is also associated with $dS_{aO_2}s$ and sleep disruption. DESIGN: Twenty-seven postpolio ventilator-assisted individuals (VAIs) using mouthpiece IPPV with little or no ventilator-free breathing time (VFBT) underwent nocturnal oxyhemoglobin saturation (S_{aO_2}) monitoring. In addition, 15 underwent nocturnal capnography and 13 underwent polysomnography. RESULTS: Mean nocturnal S_{aO_2} was normal in 22 of 27 and maximum end-tidal P_{CO_2} was normal in 12 of 15 VAIs. Use of lipseal retention for nocturnal mouthpiece IPPV significantly improved blood gas values during sleep. The polysomnography results demonstrated relatively normal sleep efficiency. CONCLUSIONS: Nocturnal mouthpiece IPPV is most effective with lipseal retention. It can provide normal alveolar ventilation and S_{aO_2} during sleep for VAIs with little or no measurable vital capacity or VFBT. Because transient $dS_{aO_2}s$ can be eliminated with lipseal retention, it may disrupt sleep less than nasal IPPV.

A Novel Mutation in the Cystic Fibrosis Gene in Patients with Pulmonary Disease but Normal Sweat Chloride Concentrations—WE Highsmith, LH Burch, Z Zhou, JC Olsen, TE Boat, A Spock, et al. N Engl J Med 1994;331:974.

BACKGROUND: Many patients with chronic pulmonary disease similar to that seen in cystic fibrosis have normal (or nondiagnostic) sweat chloride values. It has been difficult to make the diagnosis of cystic fibrosis in these patients because no associated mutation in the cystic fibrosis transmembrane conductance regulator *(CFTR)* gene has been identified. METHODS: We evaluated 23 patients with pulmonary disease characteristic of cystic fibrosis but with sweat chloride concentrations in the normal range. Mutations in the *CFTR* gene were sought by direct sequencing of polymerase chain reaction-amplified nasal epithelial messenger RNA and by testing the functioning of affected epithelium. RESULTS: A cytidine phosphate guanosine dinucleotide C-to-T point mutation in intron 19 of the *CFTR* gene, termed 3849+10 kb C to T, was identified in 13 patients from eight unrelated families. This mutation was found in patients from three different ethnic groups with three different extended haplotypes. The mutation leads to the creation of a partially active splice site in intron 19 and to the insertion into most *CFTR* transcripts of a new 84-base-pair "exon," containing an in-frame stop codon, between exons 19 and 20.

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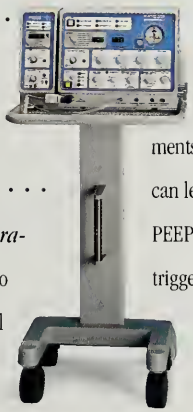

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Pulmonary Rehabilitation
By John E. Hodgkin, MD. Provides an overview of the sequence for pulmonary rehabilitation. Learn candidate evaluation and selection, rehabilitation team establishment, identification of short- and long-term goals, program components, assessment of patients' progress, and long-term follow-up. Discusses aerosol therapy, IPPB, oxygen therapy, and chest physiotherapy in the treatment of COPD. 60 minutes.
Item VT7

Sleep Apnea
By Alan K. Pierce, MD. Explains how sleep stages are related to respiratory patterns and blood gas values in both normal and abnormal subjects. Includes a discussion of the criteria for defining the sleep apnea syndrome and the distinguishing features of central, obstructive, and mixed causes of apnea. Also addressed is the efficacy of medical treatment to correct specific types of sleep apnea. 60 minutes.
Item VT11

Practical Management of ARDS
By David J. Pierson, MD. Adult respiratory distress syndrome is defined in this informative tape and its clinical features described, including incidence of risk factors and clinical predictors. Also extensively discussed is the use of PEEP to treat ARDS, including goals, complications, best or optimal PEEP levels, PEEP trials, and PEEP withdrawal, as well as general treatment, prognosis, and sequelae of ARDS. 60 minutes.
Item VT16

Pressure Support Ventilation
By Neil MacIntyre, MD. This presentation defines and describes the physiologial considerations attributed to PSV and the clinical situations when PSV may be useful. Includes comparisons when low-pressure levels and high-pressure levels of PSV are offered. Discussion includes using PSV to help overcome resistance for intubated patients and as an augmented ventilatory mode of weaning. 60 minutes.
Item VT17

Drainage of the Pleural Space: Management of Chest Tubes and Bronchopleural Air Leak
By Martha L. Tyler, RN, RRT. Learn the physiologic effects of abnormal pleural space function and the potential problems associated with chest tubes stripping, difficulties of bronchopleural air leaks with mechanical ventilation, the therapeutic goals of chest tube placement, and techniques for maintaining gas exchange with air leaks. 60 minutes.
Item VT21

Clinical Use of the Swan-Ganz Catheter
By John Marini, MD. Clinical applications of data obtained by Swan-Ganz catheter placement and the situations in which SGC placement are useful are described in this video. The clinical value of the clinical variables monitored by SGC and the complications of its placement are detailed, including "damping" of the waveform, "overwedging," and optimal lung zone placement. 60 minutes.
Item VT22

Fetal Lung Development
By Charles Rosenfeld, MD. Examines the four anatomical phases of fetal lung development along with a discussion of the biochemical development of the fetal lung through gestation. Includes a description of the substances in surfactant, the importance of their timely development, and the methods used to assess fetus survivability by using tracheal aspirant to identify the necessary ratios of the phospholipids making up surfactant. 60 minutes.
Item VT23

Theory and Application of Neonatal Ventilation
By Robert Chatburn, RRT. Knob-turning in the neonatal intensive care unit should be a profound activity because it often has profound consequences. Adjustment of a single control on a ventilator generally has multiple effects, and thorough consideration of how controls are interrelated is essential for optimum care. Presents a well-organized and systemic approach for managing mechanical ventilation. 60 minutes.
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Tissue Oxygen Delivery
By David R. Dantzker, MD. Discusses the relationship between adequate tissue oxygenation and adequate energy production, the development of lactic acidosis, and the probable role of tissue hypoxia in the multiple-organ failure of ARDS. Also discusses the factors that govern oxygen transport to the tissues and the variables that reflect the adequacy of tissue oxygen transport. 60 minutes.
Item VT26

Managing the Work of Breathing During Mechanical Ventilation
By John Marini, MD. Learn about the work of breathing and ways in which it can be minimized in the clinical setting on patients receiving mechanical ventilation. Focuses on diminishing the breathing workload through quantifying the effort during mechanical ventilation via direct measures such as oxygen consumption, electromyography, pressure-time index, and external work of breathing, as well as indirect measures. 60 minutes.
Item VT27

Pressure Support Update
By Neil MacIntyre, MD. Reviews pressure support ventilation and how it is used. Explains waveforms for airway pressure, flow, and volume. Also illustrates the measurement of inspiratory muscle loads by pressure-time index and muscle V_{O_2} and demonstrates how muscle efficiency changes as the character of work changes. Includes examples of low- and high-level PSV for elimination of the imposed work of breathing and as a weaning tool. 60 minutes.
Item VT28

The Hospitalized COPD Patient: 10 Commandments for the Clinician
By David J. Pierson, MD. Takes you inside the decision-making process of caring for a respiratory care patient with chronic obstructive pulmonary disease. Details the 10 rules for the clinician to follow when the COPD patient enters the hospital. Emphasis is on the "what not to do." 60 minutes.
Item VT29

Monitoring Respiratory Mechanics During Mechanical Ventilation
By Robert L. Chatburn, RRT. Explains how physical and mathematical models are developed and applied and how they are incorporated into ventilator design to provide estimates of mechanics. Also explains some of the problems that can develop because of the limitations of the models. 60 minutes.
Item VT30

ARDS Review
By Tony Dal Nogare, MD. Discusses the latest developments in risk factors and treatment. Also covers the five diagnostic criteria that must be present to make an accurate diagnosis of ARDS, including clinical, radiographic, and physiologic criteria. 60 minutes.
Item VT31

Sleep Disorders
By Brian Foresman, DO. A discussion of the physiology of sleep and the kinds of respiratory and nonrespiratory sleep disorders seen in the hospital. Discusses how to spot sleep apnea, the problems caused by the inpatient hospital setting, sleep disorder diagnosis, and treatment. 60 minutes.
Item VT32

Uses and Abuses of Noninvasive Monitors in Respiratory Care
By Dean Hess, MEd, RRT. Presents a discussion of how much noninvasive monitoring is needed, proof of false positives, its financial impact, and its future. Discusses the various methods: transcutaneous, pulse oximetry, and capnography. 54 minutes.
Item VT33

Nutrition and Respiratory Care
By Rich Branson, RRT. Explains the relationships and interactions of malnutrition on ventilatory drive, respiratory muscles, lung structure, and immunity. Further discusses the effects of nutrients on the respiratory system, particularly the nutritional support needed by mechanically ventilated and COPD patients. 60 minutes.
Item VT34

Smoking Cessation: Intervention Techniques for the Respiratory Care Practitioner
By Kathleen A. Smalky, MD, MPH. Covers four major aspects of smoking cessation — the impact of smoking on illness and mortality, behavioral components, current cessation programs, and effective intervention. Discusses identification of the physically addicted smoker and the effects of nicotine withdrawal. 43 minutes.
Item VT35

Hospital Acquired Pneumonia
By Galen B. Toews, MD. Provides information on the epidemiology, pathogenesis, and management of nosocomial pulmonary infections. 52 minutes.
Item VT36

New Approaches to the Treatment of Asthma
By Roger Bone, MD. This videotape discusses physician-directed adult patient education and self-management programs using a peak flow meter. Includes information on the morbidity and mortality of asthma, therapy goals from the National Asthma Education Program, and the beta-agonist controversy. Explains the "step care" treatment approach and the complications of mechanical ventilation in status asthmaticus. 60 minutes.
Item VT37

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Normally spliced transcripts were also detected at a level approximately 8 percent of that found in normal subjects. This mutation is associated with abnormal nasal epithelial and sweat acinar epithelial function. CONCLUSIONS: We have identified a point mutation in intron 19 of *CFTR* and abnormal epithelial function in patients who have cystic fibrosis-like lung disease but normal sweat chloride values. The identification of this mutation indicates that this syndrome is a form of cystic fibrosis. Screening for the mutation should prove diagnostically useful in this population of patients.

Comparison of Ballard Catheter Bronchoalveolar Lavage With Bronchoscopic Bronchoalveolar Lavage—H Levy. Chest 1994;106:1753.

BACKGROUND: Bronchoscopic bronchoalveolar lavage (BAL) in mechanically ventilated patients requires a large endotracheal tube, physician expertise, expensive equipment, and support staff. METHODS: The Ballard BAL catheter is a disposable coude tip 16F device that can be attached to the endotracheal tube and ventilator circuit without loss of positive end-expiratory pressure (PEEP) and also allows supplemental delivery of oxygen between the 12F inner and outer catheters. The catheter is directed into the selected bronchus. The inner catheter with mushroom tip is then advanced until it wedges by feel. Thirteen patients at a tertiary care, university hospital, had BAL performed through both the bronchoscope and Ballard BAL catheter using five aliquots of 20 mL of normal saline solution each. The return was quantified and submitted for blinded, paired laboratory investigations, including Gram stain and quantitative culture, and special stains and cultures as clinically appropriate. RESULTS: The procedure was well tolerated in all patients with no difference between devices in oxygen saturations; however, air leaks occurred in patients undergoing bronchoscopy and compromised safety in one. Two patients required reintubation to facilitate passage of the bronchoscope. The bronchoscopic BAL return averaged 49 mL (range, 5 to 85 mL) while BAL catheter averaged 37 mL (range, 18 to 70 mL) both being sufficient for all desired investigations except one patient who had undergone bronchoscopy. All were of excellent quality based on microscopy. The BAL results were concordant in nine patients: two *Pneumocystis carinii*, one Candida, one *Streptococcus agalactiae*, one *Streptococcus pneumoniae*, and no infection in four. Diagnoses of tuberculosis and Enterococcus (confirmed by blood culture) were obtained by the Ballard BAL catheter only. Kaposi's sarcoma and metastatic histiosarcoma were visualized by bronchoscope only. Two patients had compassionate plea use of the Ballard BAL catheter because of a small endotracheal tube. Hemorrhage secondary to lupus was documented in one and nosocomial infection was excluded in the other. CONCLUSIONS: The Ballard BAL catheter allows easy, safe BAL, without loss of diagnostic yield, when visualization is not required in mechanically ventilated patients. The Ballard BAL catheter allows maintenance of PEEP when used with the supplied adapter and can be used with small endotracheal tubes.

A Comparison of Commercial Jet Nebulizers—DT Loffert, D Ikle, HS Nelson. Chest 1994;106:1788.

Seventeen commercially available jet nebulizers from 15 commercial sources were studied (Acorn-I, Acorn-II AquaTower, AVA-NEB, Cirrhus, Dart, DeVilbiss 646, Downdraft, Fan Jet, MB-5, Misty Neb, PARI LC JET, PARI-JET, Salter 8900, Sidestream, Updraft-II, Whisper Jet). All nebulizers were filled with 2 mL of saline solution plus 0.5 mL of albuterol and powered with the same source (DeVilbiss PulmoAide). We compared total output (TO), time for total output (TTO), and percent output in respirable range (PORR). The TO was obtained by weighing before nebulization and at the point of eightfold decline in output. The TTO was calculated from initiation of nebulization to the point of eightfold decline in output. The PORR was measured by a laser particle analyzer in continuous nebulization to the same point of abrupt drop in output. The TO varied from 0.98 To 1.86 mL ($p < 0.0001$) with the Acorn-I, Acorn-II, Updraft-II, and Sidestream, significantly greater than the others ($p < 0.05$). The TTO varied from 2.28 to 20.95 min ($p < 0.0001$). The AquaTower, PARI LC JET and PARI-JET, DeVilbiss, and Dart were significantly shorter than the others ($p < 0.05$). The PORR varied from 21.89 to 71.95 percent ($p < 0.0001$). The Sidestream was significantly greater than all others ($p < 0.05$). The PARI LC JET and PARI-JET were, in turn, significantly greater than the remaining models ($p < 0.05$). To combine these characteristics, we calculated respirable particle delivery rate (RPDR) by dividing TO by TTO and multiplying by PORR. The RPDR varied from 0.03 mL/min to 0.26 mL/min ($p < 0.0001$). The PARI LC JET (0.24 mL/ min) and the PARI-JET (0.26 mg/min) had a RPDR that was significantly greater than the other models except the AquaTower, which, however, had a markedly variable performance. The Sidestream (0.19 mg/mL) did not differ significantly from the above group, nor from the DeVilbiss and Downdraft. All other models had significantly lower outputs ($p < 0.05$). We conclude that the output characteristics of commercial nebulizers vary greatly and will impact on the time required for treatment as well as the total amount of drug delivered to the lungs.

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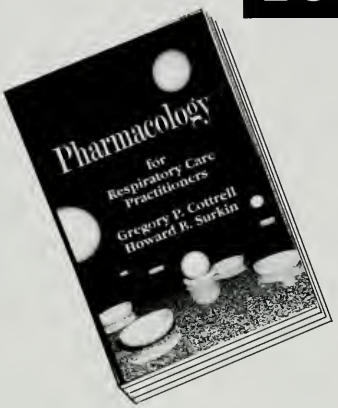

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Weaning from Mechanical Ventilation: Physician-Directed vs a Respiratory-Therapist-Directed Protocol

Gordon Wood MD, Bryan MacLeod BSc, and Susan Moffatt MD

BACKGROUND: To speed weaning and free physicians for other duties, we changed from a physician-directed to a respiratory therapist (RT)-directed protocol for weaning cardiac surgery patients from mechanical ventilation. METHODS: Eligible patients were identified by physicians after which RTs initiated and directed an intermittent mandatory ventilation weaning in accordance with a written weaning protocol. Eligibility criteria for entry into the weaning protocol (Phase 1) were expanded at the end of 4 months to include more complicated and unstable patients to be weaned by the RT-directed protocol (Phase 2). Total ventilation time and the duration of weaning were recorded for all patients during both phases and during the 2-months prior to implementing RT-directed weaning (control period). All patients were physician weaned during the control period. RESULTS: No complications were associated with the introduction of RT-directed weaning. The percentage of all cardiac surgery patients weaned by the RTs increased progressively from 41 to 90% over the 7-month study period. When compared to a similar group of physician-weaned patients in the control period, the RT-weaned patients in Phase 1 and Phase 2 of the study had a significantly shorter median total ventilation time (18.6 vs 16.8 hours [$p = 0.02$, $\chi^2_{.95,1}$] for Phase 1 and 19.7 vs 17.8 hours [$p = 0.04$] for Phase 2). CONCLUSIONS: We have demonstrated that respiratory therapists can safely and efficiently wean cardiac surgery patients from mechanical ventilation. [Respir Care 1995;40(3):219-224.]

Background

Cardiac surgery patients make up a large proportion of patients admitted to some intensive care units (ICUs). It remains the convention in many centers to use a high-dose narcotic anesthetic and to ventilate patients for 4 to 12 hours after surgery.[1] Depending on the ICU, weaning is the responsibility of the physician, nurse, or respiratory therapist (RT). In university-affiliated ICUs, the physician is often a resident in training.

It had been the practice in our unit to have the ICU resident direct the weaning process. The RT's responsibility was to make the ordered ventilator changes, not to initiate them. To speed weaning and free up physicians for other duties, we proposed to have the RTs initiate and carry out the weaning of cardiac surgery patients from mechanical ventilation. Although many ICUs have RT-directed weaning, a suitable published protocol to use as a guide in switching from a physician to an RT-directed weaning could not be found in the literature. One previously published protocol[2] showed that RT-guided weaning shortened the duration of ventilation and weaning in uncomplicated cardiac surgery patients. However, this study used T-piece weaning and the our unit has used intermittent mandatory ventilation (IMV) mode for weaning. Although neither technique has been shown to be superior to the

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The study had no outside sources of support, and the authors have no financial relationships with commercial products or interests.

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other,[3] we selected IMV as the method of weaning so that the changes involved when transferring responsibility for weaning from physicians to RTs were as few as possible. Therefore, we designed a protocol for RT-directed IMV weaning from mechanical ventilation.

This paper describes the protocol and documents its initiation and evolution. The effectiveness of our RT-directed weaning protocol is evaluated by comparing it to the previously used physician-directed weaning.

Methods

The study was conducted in accordance with the standards of the Hospital Ethics Committee who approved the study but waived the requirement for informed consent. The study was carried out in the intensive care unit (ICU) at the Kingston General Hospital. This is a 15-bed medical and surgical ICU that admits 1,200 patients/year including 450 cardiac surgery patients. The unit is staffed by 4 resident physicians who rotate through the ICU from medicine, anesthesiology, surgery, and emergency medicine training programs; these rotations last from 2-6 months and the residents are supervised by an attending intensivist.

Patient Management

No change in surgical or anesthetic technique or in patient selection occurred during the study period. The only change in ICU management involved the introduction of the RT-weaning protocol. Patients undergoing cardiac surgery are given a high dose fentanyl anesthetic (30-50 μg/kg), and all have pulmonary-artery catheters inserted. They are transferred to the ICU following surgery and are initially ventilated using the IMV mode at a rate of 10 breaths/min, a tidal volume (V_T) of 10 mL/kg, and a positive end-expiratory pressure (PEEP) of 5 cm H_2O. On admission, the F_{DO_2} is set at 0.50 (unless otherwise requested by the anesthesiologist) and initially adjusted to keep the oxygen saturation as measured by pulse oximetry (S_{pO_2}) > 95%. The first arterial blood-gas and pH analysis (ABG) is measured 20 minutes after admission, and the respiratory rate is adjusted to maintain a P_{aCO_2} of 35-45 torr. The F_{DO_2} is adjusted to keep the P_{aO_2} 70-100 torr up to a maximum F_{DO_2} of 0.60 after which the PEEP and/or the F_{DO_2} are increased to maintain a P_{aO_2} > 70 torr. Weaning from mechanical ventilation is started once the patient is warm, hemodynamically stable, awake, and obeying simple commands. This normally occurs 8-16 hours following admission in uncomplicated patients. Weaning is facilitated by reducing the IMV rate followed by a T-piece trial and extubation. Introduction of the RT-directed weaning protocol did not change the initial clinical (including ventilatory) management of these patients in the ICU or the approach to weaning. The goal was to have the patient extubated and ready for discharge to a step-down unit the morning following surgery.

Patient Selection for RT-Directed Weaning

Table 1 lists the criteria that excluded patients from the weaning protocol. Excluded patients continued to be physician weaned. Patients eligible for the RT-directed weaning protocol included all stable elective and emergency coronary artery bypass (CABG) patients. Patients with an intra-aortic balloon pump (IABP), patients who had undergone previous cardiac surgery (re-do), and patients who had undergone cardiac valve surgery were not eligible during the first 4 months of the protocol (Phase 1). Because no problems occurred with the introduction of the protocol, the eligibility criteria were expanded to include these three groups in the last 3 months of the study (Phase 2). The protocol did not change between Phases 1 and 2; only the eligibility criteria differed.

Table 1. Exclusion Criteria for the Respiratory-Therapist-Directed Weaning Protocol

Phase-1 exclusions were patients undergoing
Repeat coronary artery bypass graft (CABG)
Intra-aortic balloon pump assistance
Cardiac valve surgery or combined CABG/valve surgery
Exclusions on admission to the intensive care unit (ICU) were patients with
Major intra-operative problem
Difficult intubation
Exclusions due to unstable initial ICU course were patients with
Cardiac index < 2.5 L · m^{-2} · min^{-1}
Bleeding (> 1 L in 6 hours)
Uncontrolled ventricular arrhythmia requiring infusions of antiarrhythmic drugs
F_{DO_2} > 0.6 or PEEP > 5 cm H_2O required to maintain a P_{aO_2} > 70 torr
Need for inotropes (> 3 μg · m^{-2} · min^{-1} of dopamine or use of any other inotrope)
Oliguria (< 0.5 mL/kg for more than 2 hours)

After admission to the ICU, patients with an initially unstable course were excluded based on criteria outlined in Table 1. The residents were requested to assess the patient for eligibility for the RT-directed weaning protocol between 4 and 8 hours after admission. Transient instability that had resolved by this time did not exclude the patient from being eligible for RT weaning.

RT-Directed Weaning Protocol

Eligible patients were identified by the resident and if none of the exclusion criteria were met, an order was writ-

ten to wean the patient according to the RT-directed protocol as described in Table 2. The actual timing of the initiation of weaning was at the discretion of the patient's nurse and the RT. Weaning was to begin when the patient had regained consciousness from the anesthetic, was obeying simple commands, and was hemodynamically stable. The speed of progression of weaning was determined by the RT. The only restriction was that the IMV (breathing rate) could not be reduced more than 6 breaths/min at one time or by more than 6 breaths/min in a single hour. There was no minimal amount the patient could be weaned and no restriction on the time between ventilator-setting changes other than those mentioned previously.

Table 2. Respiratory-Therapist (RT)-Directed Weaning Protocol

- The physician's written order identifies the patient as eligible for the RT-directed weaning protocol.
- Timing of the initiation of weaning is at the discretion of the nurse and RT.
- All patients must have continuous oxyhemoglobin saturation monitoring by pulse oximetry (S_{pO_2}), and the S_{pO_2} must be within 5% of that determined from the arterial blood gas-pH (ABG) analysis done on admission and 4 hours later.
- ABGs drawn at the start of weaning must reveal a $P_{aO_2} > 70$ torr and a $P_{aCO_2} < 50$ torr on a $F_{DO_2} < 0.6$ and a PEEP ≤ 5 cm H_2O.
- When the patient is awake, obeying simple commands, and stable, the RT may reduce the breathing rate but
  no more than 6 breaths/min at one time and
  no more than 6 breaths/min in a single hour.
- If none of the weaning failure conditions are present (ie, $S_{aO_2} < 90\%$, $P_{aO_2} < 70$ torr, $P_{aCO_2} > 50$ torr, diaphoresis, spontaneous respiratory rate > 20 breaths/min, agitation or decreased level of consciousness, heart rate increase > 20%, blood pressure change > 20%, cardiac output reduction > 30%, or ventricular arrhythmias), the breathing rate is reduced and the patient is placed on a T-piece for a minimum of 20 minutes prior to extubation.
- If the T-piece trial is well tolerated (ABG values are within prespecified limits) and the physician's exam is unremarkable, the order to extubate the patient is given.

The protocol mandated that an ABG was to be measured at the start of weaning, after each ventilator change, prior to extubation, and 1 hour after extubation. However, additional ABGs could be taken at the nurse's discretion. The RTs were instructed to stop weaning, draw an ABG, and increase ventilator support, if the patient exhibited any of the failure-to-wean criteria listed in Table 2. The protocol only required the physician to assess the patient if increasing the ventilator support did not resolve the problem. Once the patient was stabilized, the RTs could again start weaning without having to consult the physician. Patients were classified as protocol failures and were physician weaned if they repeatedly failed weaning or remained unstable despite the resumption of mechanical ventilation.

Data Analysis

To determine the effectiveness of the RT-directed weaning protocol, the following data were collected on all patients; the time from admission to extubation or total ventilation time (TVT), the weaning duration (WD), whether the patient was weaned by physicians or by the RT-directed protocol, and whether the patient failed RT-directed weaning and had to be physician weaned. The WD was taken as the time from the first reduction of the IMV rate until extubation. If the subject failed an initial weaning attempt requiring the resumption of ventilation, the beginning of weaning was defined as the start of the initial attempt to wean, for the purposes of calculating WD. This information was recorded on a prepared form by the patient's nurse at the bedside. All the charts were reviewed in a blinded fashion to determine the in-ICU and in-hospital mortality and to determine whether any of the study patients required reintubation within 24 hours of extubation. Data were collected for a 2-month period prior to initiation of the protocol to serve as a historical control group. Although all the patients during this control period were physician weaned, it was prospectively determined whether they met the criteria for RT-directed weaning. This determination was made by the ICU physician prior to the start of weaning.

Patient age is presented as the mean ± (SD). When comparing the demographics of the control, Phase-1, and Phase-2 patients, discontinuous data were analyzed using a simple χ^2 statistic and continuous data with one-way analysis of variance. The results of the TVT and WD had a skewed distribution, so the medians (interquartile range) tile presented as the measures of central tendency, and the Mann-Whitney U test and Kruskal-Wallis analysis of variance were employed to evaluate these data. Statistical significance was set at $p < 0.05$.

Results

Data were collected for 9 months including the 2-month control period, the 4-month Phase-1 period, and the 3-month Phase-2 period. There were no major problems encountered during the change from physician-directed to RT-directed weaning. Over the 9-month period, 4 patients required reintubation within 24 hours of extubation—2 physician-weaned patients from the control period and one physician- and one RT-weaned patient from Phase 1. All of the RT-weaned patients survived to be discharged from the ICU. Seven patients (4%) met the eligibility criteria for RT weaning but were protocol failures and eventually physician weaned—4 from Phase 1 and 3 from Phase 2. These patients were included in the RT group for the data analysis (intention to treat).

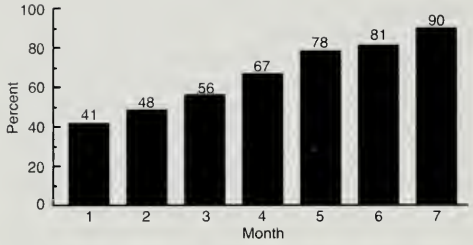

Fig. 1. Percentage of all cardiac surgery patients weaned by RT protocol during each month of the study.

There was a progressive increase in the proportion of patients weaned by the RTs during the protocol period, reaching 90% of all cardiac surgery patients by the seventh month (Fig. 1). The control, Phase-1, and Phase-2 patients were not different in terms of demographic characteristics (Table 3).

Table 3. Demographic Characteristics of the Cardiac Surgery Patients

Characteristic	Control Group	Phase-1 Patients	Phase-2 Patients
n	75	122	87
Age [mean (SD) years]	60.6 (9.7)	60.5 (12.5)	59.2 (11.9)
Gender (M/F)	56/19	93/29	69/18
CABG *(%n)	58 (77)	95 (78)	70 (80)
Isolated valve (%n)	7 (10)	15 (12)	12 (14)
CABG and valve (%n)	10 (13)	12 (10)	5 (6)
IABP (%n)	11 (15)	24 (20)	10 (11)
Redo Surgery (%n)	3 (4)	4 (3)	3 (3)

*CABG = coronary artery bypass graft; IABP = intra-aortic balloon pump.

Figure 2 shows the distribution of the TVT for all cardiac surgery patients during the control, Phase-1, and Phase-2 periods. Because there is an unequal number of subjects in each Phase, the data are presented as a percentage of all subjects in each time period. This figure shows a tendency to earlier extubation of subjects in the 2 protocol periods as compared to the control period. This effect is especially evident when the proportion of subjects extubated in less than 20 hours is examined. The distribution of TVT is skewed to the right because a small number of patients in each time period required prolonged periods of mechanical ventilation. Subjects ventilated longer than 50 hours included 6 in the control period (63, 75, 101, 115, 116, and 142 hours), 10 in Phase 1 (64, 66, 67, 68, 71, 74, 93, 95, 97, and 167 hours), and 4 in Phase 2 (59, 93, 94, and 144 hours). There is a progressive reduction in the median TVT from the control period to Phase 1 and Phase 2 when all patients in each phase are included, but this effect is not statistically significant (Table 4).

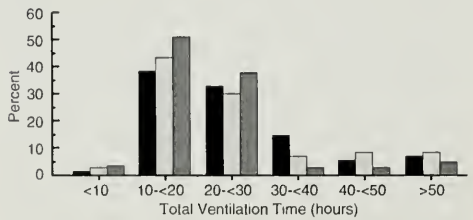

Fig. 2. Distribution of the total ventilation time (TVT) for all patients in each of the 3 study periods. ■Control (n = 75), ▩Phase 1(n = 122), □Phase 2 (n = 87).

Table 4. Effectiveness of the RT-Directed Weaning Protocol

	Control Period	Phase-1	Phase-2	p value
All Patients				
n	75	122	87	
TVT*	21 (17.8-30.6)	20.4 (16.0-26.0)	18.9 (15.9-21.9)	0.09
WD	3.3 (1.8-8.0)	3.1 (1.9-5.6)	2.9 (1.7-5.6)	0.77
Patients Meeting Phase-1 Criteria for Weaning Protocol				
n_1 (%n)	35 (47)	55 (45)		
TVT	18.6 (17.2-21.7)	16.8 (13.5-20.8)		0.02
WD	3.0 (1.5-4.2)	2.2 (1.9-4.0)		0.59
Patients Meeting Phase-2 Criteria for Weaning Protocol				
n_1 (%n)	51 (68)		70 (80)	
TVT	19.7 (17.6-22.4)		17.8 (15.6-20.8)	0.04
WD	3.3 (2.3-5.4)		2.7 (1.8-4.2)	0.48

*TVT = total ventilation time (hours) and WD = weaning duration (hours). TVT and WD data are median (interquartile range).

Figure 3 shows the distribution of the WD for all patients in the three study time periods. It demonstrates the tendency for more of the Phase-1 and Phase-2 patients to have a WD of under 4 hours compared to control-period subjects. The median WD when all patients were included decreased from 3.3 hours in the control period to 3.1 hours in Phase 1 and 2.9 hours in Phase 2 of the study (Table 4). This effect was not statistically significant. A number of patients in each phase had a WD of greater than 10 hours. This was because the WD was taken as the time from the first reduction of the IMV rate until the patient was extubated. On some occasions, patients failed the first attempt

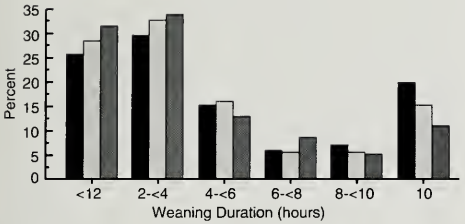

Fig. 3. Distribution of the weaning duration (WD) for all patients in each of the 3 study periods. ■Control (n = 75), ▩Phase 1(n = 122), □Phase 2 (n = 87).

at weaning and required reventilation for a number of hours before another attempt was made.

When only the RT-weaned patients in Phase 1 are compared to control patients meeting the Phase-1 eligibility criteria for protocol weaning (determined prospectively), a significant reduction in TVT is found (Table 4). Although there is a reduction in WD, it is not statistically significant. Because of the Phase-2 expansion of eligibility criteria, the group of patients weaned by the RT-directed protocol in Phase 2 consisted of patients who were clinically more complex and who would be expected to have longer ventilation times compared to the RT-weaned patients in Phase 1. This was, in fact, the case. The control group was retrospectively analyzed to create a group of patients who would have met the Phase-2 criteria for RT-directed weaning protocol. This group included all those control-period patients eligible for the Phase-1 protocol in addition to valve, re-do, and IABP patients who met none of the other exclusion criteria listed in Table 1. When this group of control patients is compared to those weaned by the RT-directed weaning protocol in Phase 2, a reduction in TVT and WD is found (Table 4). As with the Phase-1 group, only the reduction in TVT is statistically significant.

Discussion

A successful transition from a physician- to RT-directed weaning from mechanical ventilation was made with no major complications. The primary goal of the process was to transfer the responsibility for this time-consuming job to the RTs so that physicians could use their time to deal with more critically ill patients. This was successfully accomplished. There was a progressive increase in the percentage of patients eligible so that by the seventh month 90% of all elective and emergency cardiac surgery patients were being weaned by the RTs. Some of this increase was due to the expansion of the eligibility criteria that occurred at the end of the fourth month. Because all those involved had become experienced with the weaning protocol and because it had been introduced without complications, a decision was made to include the potentially more unstable re-do CABG, cardiac valve, and IABP patients. It is likely that not all of the increase in protocol use was due this expansion of the eligibility criteria. Of the control patients, 68% met the Phase-2 criteria for the RT-directed weaning protocol, whereas 80% of the Phase-2 patients were weaned by the RTs. Assuming that the patient population did not change, this suggests that there was a tendency to bend some of the eligibility criteria to allow progressively more of the borderline patients to be weaned by the RTs. Because there was no set time to determine whether patients should be excluded due to an initially unstable ICU course, patients who were initially on $F_{DO_2} > 0.60$ or those requiring inotropes were often followed longer and entered into the protocol after their inotrope and oxygen requirements had decreased. When the protocol was first implemented and when the patients in the control period were being assessed, patients were excluded earlier and not given a chance to improve.

A secondary objective of this protocol was to speed the process of weaning and to shorten the length of ventilation and this was achieved. The RT-weaned patients had a significantly shorter duration of ventilation compared to a group of historical control patients who were similar to study patients but were weaned by physicians. When all cardiac surgery patients, even those weaned by physicians, were included, the median length of ventilation decreased by approximately 2 hours from the control period to the last 3 months of the protocol (Phase 2). The reduction in TVT in the RT-weaned patients was due both to a quicker initiation of weaning and to a faster rate of weaning, once started. The protocol was not strict about when weaning could begin. This allowed the RTs to reduce TVT by initiating the weaning process earlier.

There may be many explanations for the improved efficiency of the RTs. The implementation of a fixed protocol itself may have had an effect. Weaning protocols have been shown to decrease ventilation times in studies where there was no change in personnel responsible for weaning.[4,5] During the initial part of their ICU rotation some residents are conservative in their approach to weaning due to a lack of experience. Having the patients extubated and ready for discharge to a step-down unit the next morning required that the patients be weaned during the night when the resident was the only physician on site. RTs are present in the ICU around-the-clock and, therefore, are capable of providing a consistent standard to the weaning of patients. The experience they gain remains in the ICU unlike with the residents who move on just when they become proficient in ventilator management. The progressive reduction of ventilation times from Phase 1 to Phase 2 illustrates the effect of that experience.

An additional advantage of using RT-directed weaning relates to the more focused nature of RTs' duties. The RTs can begin the weaning and continue it on their own if the physician is busy with other unstable patients. This may partly explain the more aggressive strategy employed by the RTs in initiating the weaning process sooner. We did not analyze the financial implications resulting from the reduction in ventilation time, but we assume that a quicker extubation and discharge from the ICU is a cost benefit to the hospital.

One potential limitation of this study is that the effectiveness of the weaning protocol is determined by using a historical control group that was physician weaned. It is difficult to be sure that the improvement in TVT and WD is due to the RT-directed weaning protocol and not due to unidentified changes in the patient population or patient management that occurred between the control and protocol periods.

The success of any protocol can be judged on the incidence of re-intubations because it has been demonstrated that failed extubation increases morbidity in ICU patients.[6] Only one RT-weaned patient required reintubation within 24 hours of extubation, and this patient was successfully extubated and discharged from the ICU 2 days later. The RTs only had to relinquish the responsibility for weaning 4% of the patients they started because of repeated failures to wean. This compares favourably with the study by Foster et al[2] in which 17% of CABG patients entered into an RT-directed weaning protocol were eventually physician weaned. Our protocol was designed so that patients would not fall out automatically if they failed to weaning on the first attempt. Residual effects from the high-dose narcotic anesthetic can lead to CO_2 retention and an initial failure to wean. If these patients are reventilated they often successfully wean later once the narcotic effects have dissipated. Further attempts to shorten the ventilation times in our patients will likely be limited by the anesthetic technique. Inhalation anesthesia, with its quicker emergence, has been shown to reduce the need for postoperative mechanical ventilation in cardiac patients, whereas high-dose narcotic techniques require it .[7,8]

We conclude that respiratory-therapist-directed weaning of cardiac surgery patients, following a protocol and under the supervision of physicians, can be safely used in the ICU. In our institution it reduced ventilation times and improved the efficiency of the unit with a potential cost benefit to the hospital.

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CORRECTION

Because of errors recently noted in Table 4 from Stoller JK. Travel for the technology-dependent individual. Respir Care 1994;39(4):347-362, we reprint the table in its entirety. We regret the errors.

Table 4. Equations for Predicting P_{aO_2} at Altitude*

First Author	$P_{aO_2\ altitude}$ =	r†
Henry[23]	$(20.38) - (3.0)\,CA + (0.67)\,P_{aO_2\ ground}$	0.83
Gong[6]	$22.8 - (2.74)\,Alt = (0.68)\,P_{aO_2\ ground}$	0.94
Dillard[25]	$0.453\,(P_{aO2\ ground}) = (FEV_1\ \%\ pred) + 2.440$	0.85
Dillard[26]	$P_{aO_2\ ground} \cdot e^{[(0.01731 \quad 0.000119)(FEV_1\ \%\ pred)][P_{IO_2\ ground} \quad P_{IO_2\ altitude}]}$	0.92

*CA = cabin altitude (ft); $P_{aO_2\ ground} = P_{aO_2}$ at ground level; Alt = altitude in thousands of ft; $P_{aO_2\ altitude} = P_{aO_2}$ at altitude; P_{IO_2} = oxygen tension in inspired air.
†Correlation between measured and predicted P_{aO_2} at altitude.

New Horizons in Respiratory Care: The First Decade

This issue of RESPIRATORY CARE contains 11 articles developed from the 10th annual symposium, New Horizons in Respiratory Care, which took place last December during the AARC Annual Meeting in Las Vegas. Publication of these articles marks an important anniversary and provides an opportunity to comment briefly on the history of an important feature of both the Annual Meeting and the Journal.

An idea for a new, different kind of symposium was presented to the AARC's Program Committee at its February, 1985, meeting to plan that year's convention. This new symposium would serve two purposes--to address current, unsettled issues affecting clinical practice and to generate papers on these issues for RESPIRATORY CARE, thus providing access to background information and perspective not otherwise available to the Journal's readers. The theme of What's New in Respiratory Care? was selected, sponsorship for speakers and publication was obtained from the Schering Corporation, and the first New Horizons Symposium was held in Atlanta the following November.

So successful were the 5 presentations at this symposium and the papers that followed that plans were immediately made to repeat the process the following year. Since that time, the Symposium has become a focal point of each AARC Annual Meeting, and the articles it generates are acknowledged as valuable, often unique resources for the clinician. Supported for the first 6 years by Schering, the Symposium has been sponsored by Lifecare since 1991.

The New Horizons topics and the way in which the Symposium's speaker/authors approach them are different from those in other symposia and review articles. Selected topics must fulfill several criteria: they must be 'hot,' exerting an effect on the respiratory care community, and of concern to a broad audience; they must be clinically relevant, in a readily foreseeable future if not already in day-to-day practice; and they must be either controversial or sufficiently new that definitive published literature is unavailable. Once the topics are decided upon, speaker/authors are selected who also meet specific criteria: they must be recognized authorities in the general field if not specifically in the subject at hand; they must have the ability to summarize and synthesize an extensive, contradictory, or incomplete database; and they should either have no personal connection with the topic under discussion or have a reputation for objectivity and open-mindedness.

The charge given to each presenter is a specific and often difficult one. In the presentation and also in the manuscript, the speaker should first give relevant background on the topic and define as precisely as possible what the problem or contention is. He or she should then provide a concise summary of available studies or other data pertaining to the subject (ie, the current state of the art) followed by an interpretation or synthesis of the issue (ie, what it all really means to the clinician). Finally, each speaker is charged with providing the audience and, subsequently, the reader with recommendations on how to approach the problem or deal with the issue, based not only on the available data but also on the speaker's personal experience and perspective.

Over the years, the New Horizons speaker/authors have been a diverse group, and they have done a remarkably good job with these often difficult tasks. The speakers and primary authors include respiratory care practitioners, pulmonologists, anesthesiologists, surgeons, pediatricians, biomedical engineers, a nurse, and an attorney. In the 10 years of its existence, the Symposium has generated 68 original articles and 12 accompanying editorials for the Journal. These are listed in order of publication at the end of this text. The articles themselves have ranged from 4 to 18 published pages, with a mean of 8.3 pages, providing on average nearly 60 pages of text for the Journal's readers each year. Often these papers have turned out to be the definitive reviews on their particular topics; a number have become regular handouts and supplementary teaching materials; and several of them have been widely cited in other publications. At least a half-dozen of them remain the only published discussion on their particular topic.

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As the New Horizons series enters its second decade, the field of respiratory care faces a broader spectrum of issues than ever before. If the successes of the first 10 installments in the series can be achieved by those that follow, New Horizons in Respiratory Care will continue to make important contributions to the science and practice of respiratory care in the years to come.

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The New Horizons Papers, 1986-1995

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Weaning from Mechanical Ventilation: Why All the Confusion?

David J Pierson MD

I. Weaning Is Not the Same Thing in All Clinical Settings
II. Problems with Existing Studies on Weaning
III. What is Weaning?
IV. Does Weaning Technique Really Matter?

Weaning Is Not the Same Thing in All Clinical Settings

Weaning from mechanical ventilation is part of the 'bread-and-butter' of respiratory care—something that physicians, respiratory care practitioners, and nurses who work in intensive care units (ICUs), post-anesthesia recovery units, intermediate care units, and other hospital settings do all the time. Thousands of patients have ventilatory support discontinued and are successfully extubated every day. Following this introduction there are 10 comprehensive articles on different aspects of weaning—can there really be that much to say about so mundane and familiar a process?

It turns out that despite the familiarity of weaning and the overall competence of many, if not most, practitioners in carrying it out, the process of removing patients from ventilators remains one of the most contentious and confusing areas of the practice of respiratory care. To illustrate some of the reasons for this, consider the following hypothetical patients:

- An otherwise healthy, middle-aged nonsmoker with normal preoperative pulmonary function tests who has been ventilated overnight following elective triple-bypass coronary revascularization.
- A frail, malnourished, 40-kg octogenarian with severe underlying chronic obstructive pulmonary disease (COPD), who has been ventilated for 3 weeks for acute respiratory failure caused by pseudomonas pneumonia and complicated by recurrent fevers and upper gastrointestinal bleeding.
- A 24-year-old, 120-kg football player with acute respiratory distress syndrome (ARDS) following an automobile accident in which he sustained multiple long-bone fractures and a ruptured spleen, requiring transfusions of 10 units of blood, who is now evaluated after 10 days of mechanical ventilation, receiving F_{DO_2} 0.5 and 10 cm H_2O of positive end-expiratory pressure (PEEP), with a total minute ventilation ($\dot{V}_E$) requirement of 20 L/min and a respiratory system compliance of 20 mL/cm H_2O.
- A young motorcyclist with a severe closed head injury, still unresponsive with elevated intracranial pressure after 4 days of mechanical hyperventilation, with P_{aCO_2} 25 torr and pH 7.46.
- A middle-aged patient with facial burns and smoke inhalation, sustained 24 hours ago in a house fire, who is awake and alert; her spontaneous ventilatory mechanics are good and her arterial blood gases much improved on synchronized intermittent mandatory ventilation (SIMV), with F_{DO_2} 0.35 and $\dot{V}_E$ 8 L/min.
- A college student with Guillain-Barré syndrome who has been clinically stable on assist-control for 6 weeks.

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These imaginary patients and their circumstances are very different, and they present very different considerations, when it comes to weaning. The first patient, with neither acute nor chronic lung disease and a need for ventilatory support that has now resolved, should be able to be weaned and extubated without difficulty, regardless of the predictive measurements or withdrawal technique employed. Patient 2, however, with severe underlying illness and several active acute problems, would likely fail any approach to weaning. Similarly, although he was in good health before his accident, Patient 3 remains critically ill and unlikely to be able to keep up with the demands on his ventilatory apparatus for very long. These last two patients will pose significant, very different problems in weaning even after their acute processes have improved.

For the fourth patient, mechanical hyperventilation is a specific therapeutic intervention to reduce intracerebral blood volume and intracranial pressure,[1] and weaning would not seem advisable at present. Even after intracerebral edema has resolved, discontinuation of ventilatory support will first involve cautious reversal of the respiratory alkalosis prior to any consideration of withdrawal and extubation.

Patient 5 poses a weaning problem—or, rather, an extubation problem—different from the others. Most likely, this patient could ventilate and oxygenate without difficulty off the ventilator, but with facial burns and smoke inhalation she has a high likelihood of upper airway injury, and might be unable to breathe without the endotracheal tube. Clinical assessment in this circumstance must include evaluation of upper airway patency[2] in addition to pulmonary mechanics and gas exchange.

The sixth patient presents yet another scenario—one that is distinct, in the setting of prolonged mechanical ventilation in neuromuscular disease. In this patient, consideration must be given not only to ventilatory function, gas exchange, secretion clearance, and upper airway protection, but also to psychological factors, to available techniques and devices for partial or intermittent ventilation, and to the possibility of ventilatory support without an artificial airway.[3]

No one would lump these 6 patients together and expect a discussion of weaning to apply equally to all of them. Someone setting out to do a study comparing two weaning approaches or protocols would want to make sure that the patients were as homogeneous as possible and would likely exclude most of the patients described here.

Table 1 lists 6 important factors that can be expected to exert major effects on the likelihood and time course of weaning and which would, therefore, need to be considered in designing a scientific study on this subject. Unfortunately, virtually every study ever published on weaning can be faulted for failing to clarify or control for at least one of these factors.

Table 1. Factors Affecting the Interpretation and Generalizability of Results from Weaning Studies

Clinical setting and reason for ventilatory support
Patient population studied—demographics and underlying disease
Duration of ventilatory support at the start of weaning
Protocol and timing used in each study arm
Definitions of weaning success and failure
Conceptual separation of weaning and extubation

Problems with Existing Studies on Weaning

An assessment to predict weaning success or failure is often made prior to an attempt to reduce or discontinue ventilatory support.[4] The weaning indices most often used are those developed by Sahn and Lakshminarayan in 1973,[5] which tend to corroborate guidelines that had been previously developed on the basis of clinical experience.[6] A spontaneous vital capacity > 10 mL/kg, $\dot{V}_E$ requirement of < 10 L/min, the ability to voluntarily double $\dot{V}_E$ during a brief maximum-voluntary-ventilation maneuver, and a maximum inspiratory pressure > 30 cm H_2O, were highly predictive of weaning success in the patients studied by Sahn and Lakshminarayan.[5] However, subsequently, a number of authors have taken issue with the value of these weaning indices, supplying data to show that they are not, in fact, accurate weaning predictors. How can this be? The answer usually lies in differences in patient population studied (typically short-term postoperative ventilation vs acute respiratory failure in COPD or surgical patients), and/or in the clinical circumstances under which the study was done.

A major addition to the time-honored weaning indices has been the rapid shallow-breathing index (f/V_T) of Yang and Tobin.[7] These investigators found that a value of f/V_T > 105 breaths · min^{-1}· L^{-1}, during spontaneous breathing but prior to any weaning attempt, was highly predictive of failure to wean, and many clinicians have added this index to their routine pre-weaning assessment. However, as happened following introduction of the earlier indices, papers are now appearing to refute the predictive value of the rapid shallow-breathing index.

One such study, by Lee and associates,[8] determined that the f/V_T cut-off of 105 breaths · min^{-1}· L^{-1} had a sensitivity of only 72% and a specificity of only 11% in predicting extubation success for 52 medical ICU patients—substantially worse than reported by Yang and Tobin.[7] Does this mean that Yang and Tobin were mistaken, and that their index is not helpful after all? Probably not. The difference is likely explainable on the basis of study design. Whereas Yang and Tobin studied all patients who were being considered for weaning, Lee and colleagues considered only patients who could successfully complete a 1-hour wean-

ing trial of continuous positive airway pressure (CPAP) or low-level pressure support, and only those who passed this trial (in the opinion of their primary physicians) were included.[8] In addition, only 9/52 patients failed weaning, as compared with 40/100 in the study by Yang and Tobin, diminishing our confidence that the differences in predictive value for weaning success and failure are valid.

The criteria selected to determine weaning success or failure can also affect a study's results. In one of the first papers on weaning with IMV, one that was later widely quoted, Downs and associates[9] determined how long it took to wean 2 groups of 12 patients using T-piece trials vs IMV. The total number of hours of ventilatory support in the T-piece group was nearly twice that of the IMV group (38 vs 21 hr). Rather than demonstrating an inherent superiority of IMV over T-piece weaning, however, this difference is probably best explained by the criteria used for determining weaning failure between the 2 techniques. Weaning proceeded in the IMV group as long as arterial pH remained above 7.30, regardless of any increase in P_{aCO_2}, but was terminated in the T-piece group if P_{aCO_2} rose by 10 torr. All patients in the T-piece group were initially hyperventilated using controlled ventilation, with mean initial arterial pH and P_{aCO_2} values of 7.49 and 32 torr, respectively, making it nearly inevitable that P_{aCO_2} would increase and that many of them would fail weaning.

As indicated in the table, the protocol and timing used in each study arm can affect the outcome of a weaning study. For example, if the protocol allows one weaning technique to proceed as rapidly as possible but restricts the rate at which support can be reduced in another technique, the first technique may appear to be superior simply as a result of the protocol rather than because it is truly superior. This may have been a factor in the recently published multi-center European weaning study, which found that patients weaned more rapidly with pressure support than by either progressive T-piece trials or a weaning regimen using SIMV.[10]

The fact that we still have no unequivocal evidence that any one weaning technique is best, despite dozens of earnest attempts, leads to two conclusions. First, it is extremely difficult to design and perform this type of clinical research. The elimination of investigator/caregiver bias is nearly impossible in a situation in which one of the protocols under investigation is used locally and considered by the people caring for the patient to be the technique of choice—in other words, better for the patient—and the inevitable dependence of the outcomes measured (typically time on the ventilator and need for reintubation) on the manner in which the study is designed. However, the second conclusion has to be that there is no best weaning technique—no single method that is intrinsically and in a clinically relevant way superior to the others. As discussed below, it is likely that any of several approaches can be used effectively so long as it is used correctly.

What Is Weaning?

Beyond the controversies about how best to predict that a patient can safely have ventilatory support withdrawn and which technique, if any, is best for doing this, there is confusion about how weaning should be defined. Everyone would agree that a patient who has come off the ventilator, been extubated, and transferred out of the ICU the following day has been weaned. But what about a patient who remains intubated but whose ventilatory support has been reduced to 5 cm H_2O of pressure support and 5 cm H_2O of CPAP? Or a patient breathing on a T-piece for one hour? Or for 4, or 12 hours? What about the patient who has been extubated but is still receiving positive pressure ventilation by nasal mask? What if the patient is only on nocturnal CPAP? The issue becomes more and more sticky as specific examples are considered.

This is not just a problem for individual clinicians in deciding when a patient has been successfully weaned. It is one of the chief difficulties with the literature on weaning. The experts do not even agree on what weaning is. The American College of Chest Physicians Consensus Conference on Mechanical Ventilation[11] defines weaning as the gradual reduction of ventilatory support and its replacement with spontaneous ventilation. In this context, the term weaning would not even apply to the majority of ventilated patients, who can simply have the ventilator removed by whatever means the clinician chooses. Many clinicians, however, use an operational definition much broader than this, and apply the term to the discontinuation of ventilatory support regardless of the setting or time-course for this discontinuation. Even in this series of articles,[12-21] the authors vary in their definitions of weaning.

Ideas about weaning among respected authorities on the subject can vary even more widely than this. During the ACCP Consensus Conference just referred to,[11] Dr John B Downs stated flatly "...there is no such thing as weaning." He then explained that, according to the management approach he uses, all patients, at all times, receive ventilatory support at the minimum level consistent with adequate gas exchange and patient tolerance. When this minimum level is zero, the ventilator is not needed, but there is no process of deliberately discontinuing ventilatory support that begins at a particular point in time.

Some authors equate weaning with extubation and speak of extubation protocols rather than weaning protocols. In routine postoperative ventilation and some other clinical settings, both withdrawal of ventilatory support and extubation can safely be done as a single process, but in others, such as with upper airway burns in the patient example given earli-

er, separate consideration for airway protection is obviously important. The measurements derived from the studies of Sahn and Lakshminarayan[5] and Yang and Tobin[7] really assess a patient's ability to ventilate spontaneously, not the ability to maintain a patent airway or to clear respiratory secretions. Unfortunately, however, we have no extubation indices analogous to these weaning indices—ie, no quantitative measures of the adequacy of airway protection or of secretion clearance. Such indices are sorely needed.

Because ventilatory support should be initiated for specific physiologic reasons,[6,11] it makes sense to think of weaning when those reasons are no longer present. However, one could apply this concept not only to intermittent positive-pressure ventilation (IPPV), but also to PEEP, supplemental oxygen, inspired-gas humidification, and even endotracheal suctioning. Probably, in most instances, the term weaning should be reserved for the reduction and/or discontinuation of positive inspiratory pressure. Clinicians who use pressure support could make an exclusion in the case of low-level inspiratory pressure intended only to overcome the added inspiratory work of the endotracheal tube.

One way to define weaning success would be to say that some new physiologic insult would be required for positive-pressure ventilation to have to be resumed. The use of CPAP and other maneuvers to augment oxygenation would be separate. Yet, even this definition is inadequate because, as discussed in the article on weaning in long-term ventilatory assistance,[21] a patient may need IPPV only some of the time as elective therapy rather than life support. The issue remains complicated and unresolved. Nonetheless, it makes both physiologic and practical sense to think about the ventilatory management of respiratory failure not as a single process but as comprised of several distinct components:

- positive-pressure ventilation (eg, inspiratory positive pressure to take over or reduce the patient's work of breathing);
- therapy to improve oxygenation (eg, supplemental oxygen, CPAP, and other therapies to increase tissue oxygen delivery);
- provision of an artificial airway (along with humidification and warming of the inspired gases);
- facilitation of secretion clearance; and
- specific therapy for the primary disease process.

In this era of nasal ventilation and other nontraditional approaches to ventilatory assistance, it is especially important to think of ventilatory support and the provision of an artificial airway as separate processes (Fig. 1).

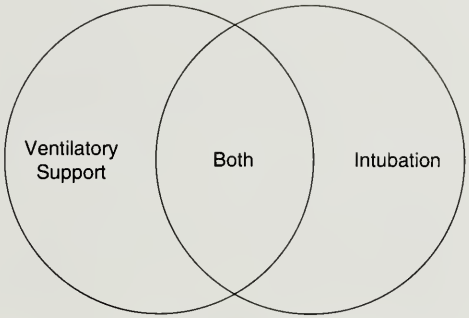

Fig. 1. Options for ventilatory support and airway access. Many patients need both positive-pressure ventilation and an artificial airway, but some need only one and not the other. Some patients can safely be weaned but not extubated, while others continue to need some ventilatory support but not an airway. A management approach based on pathophysiology and specific patient needs should consider these options as separate and distinct.

Does Weaning Technique Really Matter?

Which is better, a Ford Taurus or a Honda Accord? The two best-selling cars in the United States are comparable in size, features, performance, and price. The results of numerous comparison tests favor one or another by the narrowest of margins, or rate them a toss-up. Yet in purchasing a car the individual consumer must decide on one or the other. In this circumstance most of us choose on the basis of intangibles—experience with a particular make, the recommendations of friends, the persuasiveness of the sales staff, or simply 'gut' feelings. Both of them are excellent cars, and the great majority of drivers will be satisfied regardless of which one is selected.

The debate about which weaning approach is best is analogous to this Taurus-Accord dilemma. One study says SIMV is better; another favors pressure support; a third recommends the old-fashioned T-piece method. None of the studies is perfect, yet somehow if there were a really important difference, and one of the three were truly best in some clinically important way, one would think such evidence would have emerged by now.

As every parent of a teenager knows, adequate familiarity with the automobile's controls and safe-driving habits are far more important than whether the car is a Ford or a Honda. If one drives an automobile unwisely or with poor understanding, one can easily exceed the speed limit and cause a serious accident in either a Taurus or an Accord. Similarly, any weaning technique can be misapplied so as to make patients needlessly uncomfortable and prevent successful weaning. That one technique is selected over another is less important than that the clinician using it understands its rationale and application and applies it effectively.

Improved understanding of the weaning process and more appropriate practical application of its various tech-

niques are the focus of the 10 articles that follow this one. The reader who hopes to learn, at last, which weaning method is best will be disappointed. Instead, in these discussions the reader will find in-depth consideration of a number of facets of weaning that have been neglected or confusingly treated in previous publications. The overall purpose for the articles, as for the symposium upon which they are based, is to provide the clinician with a more complete, objective view on weaning, and to permit the use of any of the available weaning techniques in the most rational and effective way possible, while at the same time promoting the greatest degree of patient safety and comfort.

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Weaning Techniques and Protocols

John J Marini MD

I. Introduction
II. Differing Approaches to Weaning
III. Compensating for Tube Resistance and Lung-Volume Loss
IV. Pre-Extubation Testing of Spontaneous Breathing
V. Transferring the Breathing Workload
A. SIMV
B. PSV
C. T-Piece
VI. In Conclusion

Introduction

In this paper, I describe my perceptions regarding weaning, reconciling them with experience, observations, and techniques. The choice of weaning technique should be guided by the underlying reason for ventilator dependence and by sound physiologic principles. Psychological dependence, inability to adequately saturate arterial blood with oxygen, and cardiovascular instability (congestion or ischemia) are often contributory, and occasionally constitute the primary cause of weaning failure.[1,2] The most common reason for continued ventilator dependence, however, is an imbalance between ventilatory capability and demand.

Differing Approaches to Weaning

Some clinicians argue that attempts should be made to transfer to the patient as much of the breathing workload as can be tolerated from the outset of ventilatory support, so that a distinct phase of weaning does not really exist. By this reasoning, the process of discontinuing the ventilator should extend unbroken throughout the entire period of care. Others believe that attempts to transfer any substantial fraction of the breathing workload are ill-advised until the patient can easily breathe unassisted; therefore, the discontinuation of mechanical ventilation should be attempted only after the patient has fully recovered.

Many clinicians subscribe to neither of these extreme positions, but rather provide full support during an initial period, and then attempt to gradually transfer the breathing workload to the patient when signs appear favorable. My own view is that certain patients do benefit from gradual withdrawal of machine support, especially if the breathing workload or cardiac stress is disproportionate to the patient's capability to comfortably sustain it. Then, gradual weaning may allow for muscle reconditioning or reintegration of muscular coordination, which may have been lost during the period of acute illness. Gradual withdrawal of ventilatory support is also important if sudden transitions to spontaneous breathing are physiologically or psychologically stressful. Patients with such problems include those who experience panic cycles, coronary ischemia, unusually intense breathing workloads, or congestive heart failure during the weaning attempt. In this latter group, the negative intrapleural pressures that result from spontaneous breathing efforts increase both venous return and left ventricular afterload.[3] Increased oxygen consumption further adds to the cardiac workload. In essence, the weaning process is an attempt to allow the patient to adapt to otherwise adverse physiologic stresses that may be respiratory, cardiac, or psychological in nature.

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The Importance of Compensating for Tube Resistance and Lung-Volume Loss

In virtually every patient receiving partial ventilatory assistance, I use some level of pressure support (PS) and some level of end-expiratory positive airway pressure (PEEP or CPAP) during the work transferral phase. PS is the only technique currently available to help overcome endotracheal tube resistance. The level of PS required is a function of tube dimensions and inspiratory flow demand (minute ventilation divided by the inspiratory time fraction).[4] Future techniques, such as sensing airway pressure at the carinal tip of the endotracheal tube[5] or using proportional assist ventilation,[6] may avert the need for PS.

Recumbency is associated with a major loss of upright lung volume, which low levels of PEEP can help to overcome.[7] A normal subject may lose from 600 to 1200 mL of lung volume in moving from the sitting to the horizontal-supine position—a loss of volume that would normally require 5- to 10-cm-H_2O PEEP to restore.[7,8] In the clinical setting, I generally use from 3- to 7-cm-H_2O PEEP for this purpose, depending on body habitus. (Obese patients and those with a noncompliant chest wall for other reasons require the higher values). The value of adding PEEP to patients with severe airflow obstruction and auto-PEEP has been documented for ventilator-assisted breathing (Fig. 1). Adding PEEP to a patient experiencing auto-PEEP can make the breathing effort easier and facilitate coordination between patient effort and machine response.[9,10] During pressure-supported breathing, part of the patient's own effort must be used to offset any auto-PEEP that exists. Because PEEP may counterbalance (and thereby eliminate) auto-PEEP in patients with expiratory flow limitation, adding CPAP often makes a fixed level of pressure support more effective in achieving ventilation. Another reason to consider PEEP in patients with severe airflow obstruction relates to the possibility of an improved *distribution* of ventilation. It has been shown that patients with obstructive lung disease trap gas when assuming lateral-decubitus[11] or supine-horizontal[8] positions. PEEP may help reopen these airways and restore a more normal distribution of ventilation (Fig. 2).

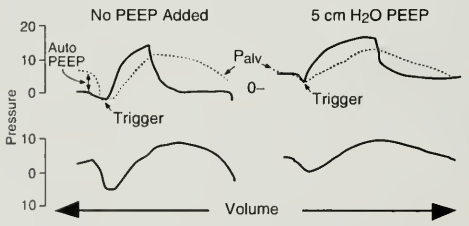

Fig. 1. Improved ventilator triggering by PEEP. Before PEEP is added, auto-PEEP must be overcome before negative airway pressure is sensed. Auto-PEEP is counterbalanced by a low level of applied PEEP.

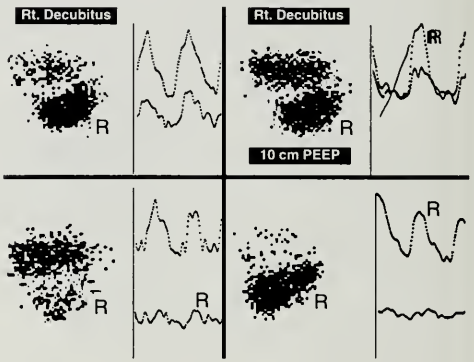

Fig. 2. Positional gas trapping and improvement with PEEP, as demonstrated by radionuclide ventilation scanning. (Normal patient, top two panels; patient with COPD, bottom two panels.) Note reversal of the normal ventilation pattern before PEEP is added, due to gas trapping in the dependent lung. Ventilation pattern is 'normalized' by the addition of 10-cm-H_2O PEEP. (From Reference 11, with permission).

Pre-Extubation Testing of Spontaneous Breathing

These important advantages of PS and PEEP underscore the importance of testing the patient's ability to breathe without such assistance immediately prior to extubation. Although PS and PEEP are useful during the period of transition to spontaneous breathing, I *do not* extubate before observing a brief trial of spontaneous breathing without the help of either of them. This practice seems reasonable because the patient is faced with loads to the respiratory system after machine discontinuation that are not present in the pre-extubation period:

- Increased work of breathing may gradually result in lung congestion in patients with cardiac disease.
- Repetitive breathing with reduced tidal volumes and reduced functional residual capacity (FRC) may result in atelectasis.
- Secretion retention may increase the work of breathing.
- Failure to swallow normally may result in aspiration.

- The tissues of the upper airway may undergo swelling and edema postextubation, which results in upper airway obstruction.[12]

For all of these reasons, the patient requires a substantial breathing reserve in the immediate postextubation period. This reserve is tested by having the patient breathe without the help of either pressure support or PEEP for a brief period prior to withdrawing the endotracheal tube.

Transferring the Breathing Workload

Given that both PS and CPAP are used during the withdrawal process on all my patients who are still attached to the ventilator, three options are available for achieving the graded transfer of the breathing workload. In conventional flow-controlled, volume-cycled synchronized intermittent mandatory ventilation (SIMV), the patient's workload is gradually increased by reducing the frequency of machine-aided breaths. In PS ventilation (PSV), each breath is ventilator assisted, but the amount of power provided per breath is reduced as pressure is withdrawn. With T-piece breathing, full ventilator support is punctuated by lengthening intervals of breathing without any machine support. Each method has its advantages and disadvantages, as well as its advocates and detractors.

SIMV

In SIMV, the primary advantages are two: first, a 'back-up' minute ventilation is guaranteed to the subject; and, second, rather large breaths are provided periodically. Some clinicians believe that the intermittent application of large tidal volumes may disturb the patient's own breathing rhythm. In my view this criticism, while understandable, may be too harsh. Many people, especially the elderly with heart and lung disease, show marked variations in tidal volume during spontaneous breathing and intermittently draw large breaths.[13] These large breaths have an uncertain function but possibly help avert progressive microatelectasis or the stimulation of dysfunctional breathing reflexes.

SIMV, however, has a number of inherent disadvantages. Perhaps the most striking one is that the patient 'uncouples' his breathing effort from the support provided by the ventilator. By this, I mean that the patient continues to make efforts of approximately equal strength, independently of whether the individual cycle in question is machine-assisted.[14,15] Moreover, available evidence suggests that once the machine's cycling frequency approximates 50% of the fully assisted (but patient-triggered) value, the patient breathes about as hard per cycle as when ventilatory support is completely withdrawn[14-16] (Fig. 3). Responses

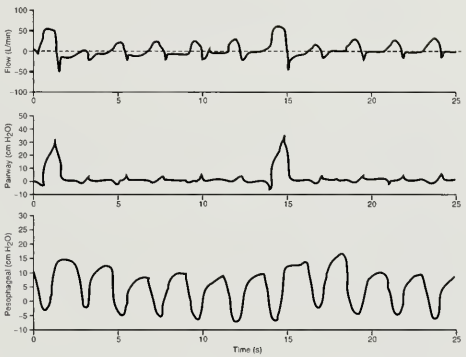

Fig. 3. Comparison of efforts during SIMV for assisted and nonassisted breaths. At any given level of machine support, the intensity of breathing effort is unaffected by airway pressurization.

of individual patients differ, and no general statement is perfectly applicable to all. However, it can be generally said that SIMV tends to reload the patient earlier in the withdrawal process than does pressure support. This early reloading is reflected in recent data comparing SIMV and pressure support in animals[17] and in patients being weaned from mechanical ventilation.[18] In a study of patients judged ready for a weaning attempt, Knebel and colleagues of our group demonstrated that breathing frequency during SIMV rose quickly as machine support was reduced from 100% to 50% of the assist/control value, but little more thereafter[19] (Fig. 4). This rapid ascent of respiratory rate during the initial phase of support withdrawal stood in marked contrast to the smooth rise in frequency seen with pressure support.

PSV

Apart from helping overcome endotracheal tube resistance, PS has the undeniable advantage of retaining the coupling between patient and ventilator throughout the entire range of its application; unlike SIMV, every breath is machine-aided. The patient also retains control over the cycle length, as well as the triggering time of the individual breathing cycle. Although it seems plausible that PS may help the patient to re-integrate the coordinated activity of respiratory muscles that is lost during the period of acute stress and pump overload, to my knowledge, no convincing evidence for this, has yet been gathered.

Although the value of PSV as a technique to gradually withdraw machine support is generally recognized, it is less well appreciated that PSV affords the patient a *flexi-*

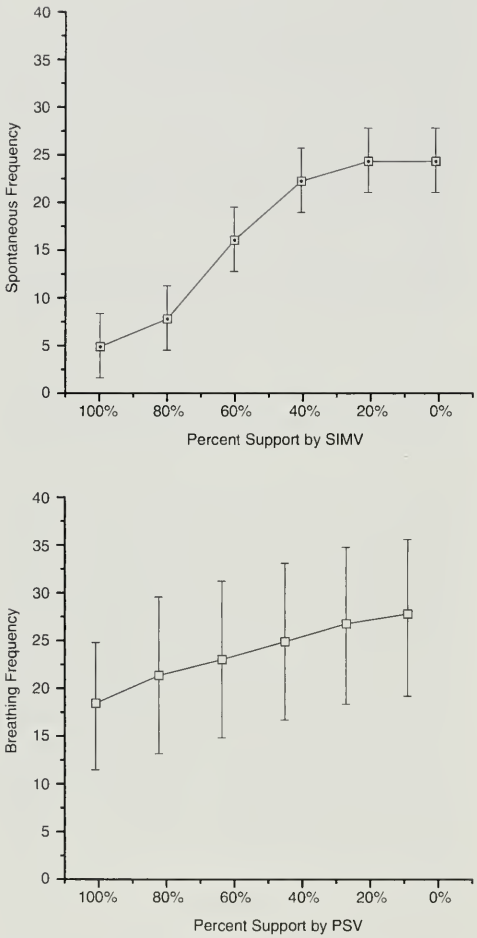

Fig. 4. Comparison of breathing frequency during withdrawal of ventilator support by SIMV and PSV. In this study, each patient was exposed to both methods of ventilator withdrawal. Note the relative abruptness of the rise in ventilatory frequency in SIMV, ⊢⊡⊣ (left), as opposed to breathing frequency during pressure support ⊢□⊣ (right). Note also the wider variability for the PSV group. (From Reference 19, with permission.)

bility in drawing power from the machine that SIMV does not.[17] If the patient chooses, additional power can be pulled from the ventilator at an unchanging level of PS simply by increasing the cycling frequency. Indeed, PSV allows a weak patient to defer reloading of the respiratory muscles until the very final stage of the weaning process. The clinical message is that physicians should be especially cautious in the earlier phases of SIMV weaning and in the later phases of PS weaning.

T-Piece Weaning

The T-piece weaning method is generally initiated with brief periods of time off the ventilator, interrupted by longer periods of full machine support (eg, assist/control). These periods off the ventilator are gradually lengthened as tolerance permits. With classic T-piece weaning, no CPAP or PS is provided, and a separate valveless external circuit is employed. This minimizes breathing resistance, but provides no airway alarms and foregoes the advantages of CPAP and tube-compensating PS already discussed. (For this reason, many clinicians prefer to employ a pressure-supported/CPAP modification of the fully spontaneous T-piece method.) One advantage of T-piece weaning is that it tests the ability of the patient to breathe spontaneously early and often, potentially speeding the process of machine disconnection. It also enables the physician to test the reserve of breathing power as the patient breathes through the endotracheal tube without PS.

As discussed earlier, a brief T-piece trial (without CPAP or PS) prior to extubation seems prudent in order to avoid delayed weaning failure in the postextubation period. Some have argued that T-piece weaning also makes good sense as a method to strengthen and train the respiratory muscles, in that stress periods are alternated with periods of near-complete rest. T-piece would seem to be a poor choice, however, for patients who are unduly stressed by *sudden* conversions between fully supported and fully spontaneous breathing. These might include patients with severe underlying coronary disease, severe muscle weakness, or those inclined toward panic reactions due to obstructive lung disease or psychological overlay.

A recently published comparative trial of SIMV, PS, and T-piece methods appeared to strongly favor PSV (Fig. 5).[20] Unfortunately, this comparative trial, although the best that has yet been conducted, leaves many questions still unanswered. In this European multicenter cooperative study, the modes were not applied as they would be clinically in most medical centers. Specifically, no machine rest was provided during sleep periods, no PS was provided for tube resistance, no CPAP was routinely employed, and the SIMV weaning began from 50% of the assist control frequency—a level associated with considerable stress, as already noted. It is interesting to note that

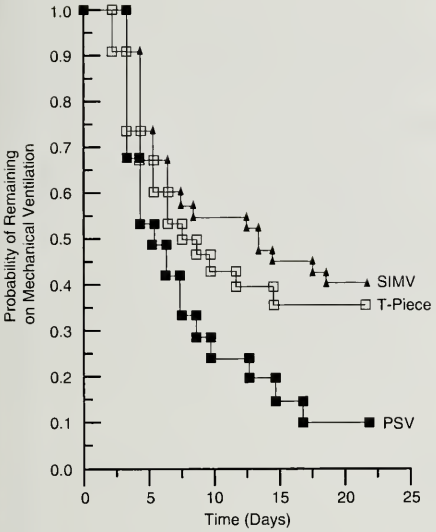

Fig. 5. Probability of remaining on the ventilator as a function of time for the three most common weaning modes. In this study, pressure support held a clear advantage. (From Reference 20, with permission).

PSV did not prove superior to SIMV in another recently published multicenter trial of weaning methods.[21]*

In Conclusion

In my view, the key factors in reducing the weaning time are (1) to withdraw support as quickly as tolerated; (2) to check the patient's ability to breathe spontaneously at least once daily; and (3) to rest the patient when overtly dyspneic and during sleep periods. The weaning priorities should be

- To maintain adequate FRC with PEEP of 3- to 5-cm H_2O.
- To minimize the breathing workload during the adaptation process with pressure support sufficient to overcome endotracheal tube resistance.
- To intermittently give large tidal breaths (10 mL/kg, at least once/minute) to keep the lung fully recruited and prevent activation of maladaptive reflexes triggered at low-lung volume.

In my practice, therefore, I provide CPAP, tube-compensating pressure support, and at least one SIMV breath/min. to all patients who require ventilatory support over extended periods. Beyond these basal requirements, some patients prefer SIMV to pressure support as the inspiratory power source, and so long as the preceding priorities are met, I am not locked into always using the same mode. If one fails I will try the other. I do not use gradually prolonged T-piece breathing as an adaptation technique. However, at least once daily and always before extubation, a T-piece trial (without any CPAP or pressure support) is mandatory to test for breathing reserve. In the immediate postextubation period, refeeding is initiated cautiously, and only after the ability to swallow fluids without aspiration has been empirically tested at the bedside under direct vision, at least several hours after extubation. Noninvasive ventilation may be extremely helpful as a bridge for marginally compensated patients to adapt to fully spontaneous breathing.[22,23]

*Abstracted on Page 206 of this issue.

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Weaning and Extubation Are Not the Same Thing

Sam R Sharar MD

I. Introduction
II. Criteria that Predict Successful Ventilation without an Artificial Airway
III. Criteria that Predict Successful Airway Patency after Extubation
IV. Summary

Introduction

In deciding whether to extubate the ventilated patient recovering from respiratory failure the clinician must consider two factors: (1) the patient's ability to provide adequate gas exchange during spontaneous ventilation, and (2) the patient's ability to provide and protect a patent upper airway. Despite the fact that a particular clinician-specialist may focus attention primarily on one or the other of these factors during the weaning process, the considerations are not mutually exclusive. In fact, weaning and extubation should be approached as two separate decision processes, both of which must be independently assessed prior to attempted extubation. For the majority of patients weaned from assisted ventilation, both issues can be satisfactorily resolved and extubation safely performed. These patients, in general, meet some consensus criteria (ie, weaning indices) that predict no further need for assisted ventilation, and *at the same time* have no anatomic or neurologic abnormalities that might prohibit a protected and patent natural upper airway. For example, the common situation is an awake, neurologically intact patient who has no upper airway abnormalities, but whose unsatisfactory values of ventilator weaning indices preclude attempted extubation. Conversely, stellar values for weaning indices may predict successful spontaneous ventilation but cannot predict the adequacy of airway patency and protection in individuals with neurologic, anatomic, or airway secretion-clearance abnormalities. Furthermore, no combination of weaning indices and patient examination can predict successful extubation with 100% sensitivity and 100% specificity. Analogous to the surgical instance in which classic signs and symptomatology of appendicitis resulting in appendectomy predict true appendicitis in only 80-85% of cases,[1] the reintubation rate for patients recovering from prolonged postoperative ventilation of respiratory failure has been reported to be 5-20%.[2-4]

Contributing to this high false-positive rate in predicting successful extubation is a subset of patients anticipated for extubation in whom either only marginal values of weaning indices are achieved or some abnormality exists that puts the airway at risk in the immediate postextubation period. In selected cases in this group, in whom maximal efforts have been made to improve gas exchange and/or eliminate airway abnormalities and for whom no further improvement is expected, extubation may still be attempted, albeit with both a high index of suspicion for failed spontaneous ventilation and advance preparation for reintubation. Many would argue that reintubation rates of up to 20% are acceptable in this subset (assuming that reintubation can be accomplished without complication), in that early extubation is more cost-effective and minimizes other complications related to endotracheal intubation and mechanical ventilation compared to unnecessarily prolonged intubation and ventilation. To minimize the rate of failed extubation, issues of both ventilator weaning and airway patency and protection are considered in order to identify patient characteristics associated with such failed attempts, focusing on criteria that predict (1) successful ventilation without an artificial airway and (2) successful airway patency after extubation.

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Criteria that Predict Successful Ventilation without an Artificial Airway

The general determinants of successful spontaneous ventilation are obvious: (1) resolution of the process for which mechanical ventilation was initiated, the presence of hemodynamic stability, and the absence of sepsis; (2) adequate oxygenation with minimal or moderate supplemental oxygen and PEEP, and (3) adequate spontaneous ventilation to maintain P_{aCO_2} near baseline. The difficulty lies in assigning specific measurable parameters of oxygenation and ventilation that are practically obtained and predict successful withdrawal of ventilatory support. In part, this difficulty is due to the many indices that currently can be measured (eg, conventional mechanical variables, endurance measurements, work of breathing, tracheal occlusion pressure, multifactorial indices), so that confusion easily arises. One major source of difficulty, however, is that clinical trials assessing the predictive success of these numerous indices have not been standardized. A variety of weaning protocols are used and widely disparate patient populations are studied, from healthy postoperative patients to the elderly with chronic respiratory failure, and from short-term to long-term ventilated patients. In addition, the definition of weaning failure or success varies among studies. Finally, because successful weaning depends on multiple physiologic systems (ie, pulmonary, cardiovascular, skeletal muscle, neurologic, and metabolic), it is likely that no single parameter can accurately represent homeostasis in all of these systems. Because of this, interpretation of published data can be confusing, and what constitutes the 'best' weaning parameters is controversial. A detailed discussion of the 'nonconventional' weaning parameters is beyond the scope of this review. However, because some of these parameters can be conveniently obtained and have been reported to be superior predictors of weaning in some cases, the reader is referred to other reviews.[2,5,6]

Table 1. Conventional Weaning Parameters

Measures of Adequate Oxygenation or Oxygen Transport
$P_aO_2 > 60$ torr with $F_iO_2 < 0.35$ on low-level PEEP
$P(A-a)O_2 < 350$ torr
$P_aO_2 / F_iO_2 > 200$
Cardiac Index > 2.1 L · min^{-1} · m^{-2}
No lactic acidosis
Measures of Ventilatory Mechanics
Vital capacity > 10 mL/kg ideal body weight
Maximum inspiratory pressure > −30 cm H_2O
Minute ventilation < 10 L/min
Maximum voluntary ventilation > 2 × resting minute ventilation
Patient-ventilator system compliance > 25 mL/cm H_2O

The conventional weaning parameters assessing gas exchange and mechanical function of the respiratory system (Table 1) are easily obtained at the bedside, have been well studied under a variety of clinical conditions,[5] and are reasonably accurate predictors of successful spontaneous ventilation, depending on the patient population.[6] Inadequate patient performance usually reflects a concurrent pathophysiologic process that should be identified and treated as effectively as possible prior to attempting extubation. Examples of the more common causes of poor performance are listed in Table 2.

Table 2. Selected Conditions Resulting in Poor Values of Weaning Indices

Oxygenation
Central hypoventilation (eg, neurologic injury, drugs)
Ventilation-perfusion mismatch due to unresolved, acute process (eg, bronchospasm, inhalation injury, congestive heart failure) or chronic, underlying pulmonary disease (eg, COPD)
Right-to-left shunt (eg, intracardiac shunt, pulmonary vasodilator therapy)
Low venous O_2 content due to excessive peripheral O_2 extraction (eg, low cardiac output, increased O_2 consumption)
Ventilation
Central hypoventilation (eg, neurologic injury, drugs)
Inadequate neuromuscular function (eg, quadriplegia, muscular dystrophy, muscle weakness due to inactivity/atrophy, increased work of breathing to overcome airflow resistance of too small an artificial airway)
Increased alveolar dead space (eg, pulmonary or fat embolism, ARDS, emphysema)
Increased CO_2 production (eg, increased O_2 consumption, particularly with carbohydrate overfeeding, fever)

The decision may be made to attempt extubation in spite of marginal values for weaning indices, assuming that personnel and equipment are immediately available for reintubation, and that the patient's condition does not increase the likelihood of complications from urgent reintubation (eg, ongoing myocardial ischemia, anticipated difficult intubation). These extubation attempts usually occur when concurrent pathophysiologic processes cannot be further improved and the only alternative is long-term mechanical ventilation. It is in these patients, in particular, that attention to minute details for optimizing spontaneous ventilation (eg, clearance of secretions, maximal bronchodilator therapy and nutrition, and optimal patient positioning to minimize loss of lung volume by extrathoracic tissues, compressing the diaphragm and chest wall) may determine success or failure at extubation.

Criteria that Predict Successful Airway Patency after Extubation

The general determinants of airway patency and protection include those related to the course of medical treatment and those related to the patient's airway anatomy and neurologic function—the common issues being airway obstruction, risk of pulmonary aspiration, and secretion clearance. For patient safety, the clinician performing the extubation should always be mentally and physically prepared to provide airway and ventilatory assistance in the event of rapid postextubation respiratory failure. In other words, skilled personnel and proper equipment for providing airway and ventilatory support should always be immediately available, with specialized equipment (eg, fiberoptic bronchoscope and needle cricothyrotomy supplies) at the bedside prior to extubation of patients with reported or anticipated difficult airways or intubations.

Extubation should not be attempted in patients with a developing medical problem that may increase the need for airway protection or ventilatory support (eg, upper gastrointestinal hemorrhage) or with impending procedures that require intubation (eg, surgical procedure with general anesthesia). Extubation in these individuals merely serves to unnecessarily increase the risk of complications associated with reintubation, particularly in those patients with difficult upper airways.

Upper airway obstruction can occur in certain individuals upon extubation regardless of the adequacy of their weaning predictors and can be a major cause of failed extubation. Severe airway edema or airway compression by external masses are the primary etiologies of such obstruction. Severe periglottic or upper airway edema can result in either complete or partial airway obstruction at extubation in patients recovering from epiglottitis, angioedema, or smoke inhalation injury. External compression of the airway with a compromise in airway caliber can occur with traumatic or postoperative hematomas in the neck, infectious masses (eg, retropharyngeal abscess), malignancies (eg, thoracic outlet syndrome), or postoperative edema following major head and neck surgery. As these processes resolve and extubation is contemplated, one method for assessing postextubation airway patency prior to extubation is the "cuff leak" test.[7] To perform this test, patients must first no longer require mechanical ventilatory assistance. The patient is then disconnected from the ventilator and allowed to breathe spontaneously through a cuffed endotracheal tube. Next, the tracheal cuff is deflated and the tube lumen manually occluded. The presence of a peritubular leak with spontaneous ventilation suggests adequate airway caliber and indicates likely success at maintaining airway patency following extubation. The cuff-leak test is easily performed and widely used and has high predictive value (100% sensitivity and 70% specificity) in otherwise healthy adults.[8]

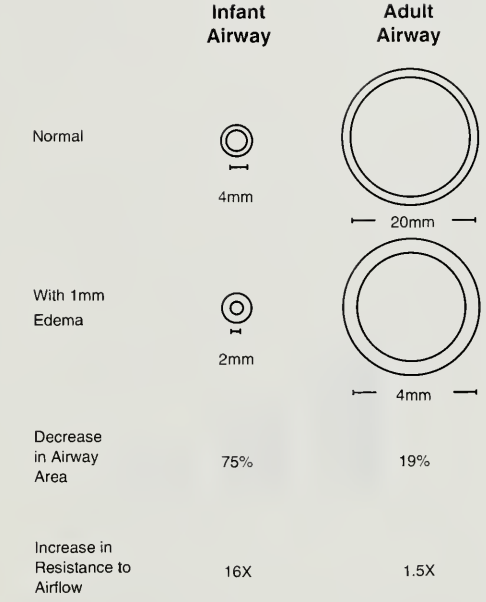

Fig. 1. The effect of 1-mm edema on internal diameter, cross-sectional area, and resistance to airflow in an infant airway compared to an adult airway. The increased resistance in the child results in audible stridor and significantly increases the work of breathing in order to maintain a given minute ventilation. (Reprinted with permission, from Reference 9).

Glottic edema as a result of endotracheal-tube-induced injury can also narrow the upper airway following extubation. The clinical consequences can vary from mild postextubation stridor, to severe stridor that interferes with ventilation, to complete airway obstruction. Severe stridor and airway obstruction are more commonly seen in children due to the small dimensions of their upper airway (Fig. 1).[9] The incidence of postextubation stridor requiring treatment in healthy children undergoing elective surgical procedures is only 4%,[10] but is reported as high as 47% in pediatric trauma and burn victims.[11] Due to the partial dependence of airflow resistance on gas density, lower density mixtures of helium and oxygen ($\geq$ 60% helium) have been shown to decrease airflow resistance[12] and reduce both the severity of stridor and the need for reintubation.[13]

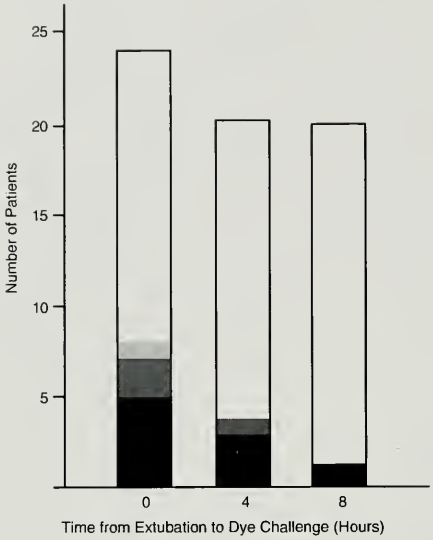

Fig. 2. Numbers of patients who aspirated radiopaque dye given orally immediately, 4 hours, or 8 hours after extubation. Patients had been intubated for 12 hours for elective coronary artery bypass surgery. ▭ No aspiration, ▭ trace aspiration, ▬ moderate aspiration, ■ massive aspiration. (Reprinted with permission, from Reference 14).

Numerous factors contribute to an increased risk of pulmonary aspiration in the recently extubated patient. First, central neurologic injury may reduce or suppress the normal gag or cough reflex that serves to protect the upper airway from the aspiration of fluids present in the posterior pharynx. Furthermore, it has been reported[14] that periglottic sensation is abnormal for 4-8 hours following extubation in patients intubated for longer than 8 hours, resulting in an increased risk of pulmonary aspiration immediately after extubation (Fig. 2). Second, mechanical closure of the glottis may be impaired following prolonged intubation, with a 28% incidence reported in those intubated longer than 6 days.[15] Resolution of this abnormality occurs over 24 hours and affects not only physical protection of the airway but also the patient's ability to generate the high intrathoracic pressures necessary to clear the airway by coughing. Finally, in a study of human volunteers given low-dose muscle relaxants, a maximal inspiratory pressure of -25 cm H_2O was associated with adequate minute ventilation but with nonfunctional muscles of airway protection (assessed by swallowing, approximation of vocal cords, ability to maintain a patent airway, and ability to elevate the mandible).[16] Airway protection was not assured until muscle strength was adequate to generate a maximal inspiratory pressure of -33 to -43 cm H_2O (Fig. 3). Therefore, for extubated patients with marginal muscle strength or marginal inspiratory effort, the risk of aspiration appears to be increased. Based on this collection of observations, fasting may be prudent in some cases (ie, discontinuance of gastric tube feedings) for 4-6 hours prior to extubation to reduce the risk of postextubation aspiration of gastric contents.

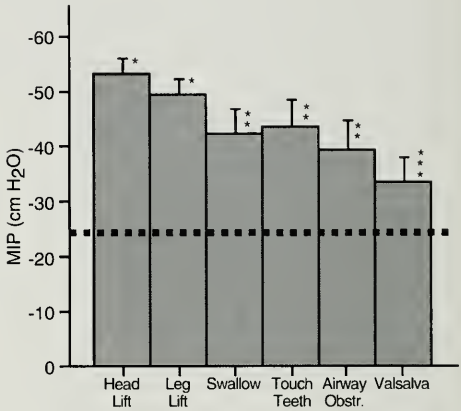

Fig. 3. Association of maximal negative inspiratory effort (MIP) with common clinical maneuvers to assess muscle strength in human volunteers receiving low-dose curare. Normal resting minute ventilation was maintained in all subjects. (Reprinted with permission, from Reference 16).

A final group of patients who may experience postextubation difficulty despite adequate weaning parameters is comprised of those who are unable to adequately clear airway secretions. These individuals include those with either inadequate neurologic (eg, anoxic encephalopathy) or muscular function (eg, mid-to high-cervical quadriplegic) to clear normal secretions, and those with excessive or tenacious secretions (eg, cystic fibrosis).

Summary

The decision to extubate any ventilated patient requires independent assessment of both ventilatory function (eg, weaning indices) and upper airway patency and protection. Although a variety of measurements can be made, no single weaning predictor is 100% sensitive and specific. A

practical approach is to assess first the conventional predictors (Table 1) and develop a consensus for the likelihood of successful spontaneous ventilation. Next, airway and neurologic examinations can help identify patients at risk for upper airway obstruction or aspiration following extubation. Combining the assessment of these two important components of weaning and extubation, Table 3 offers general, practical guidelines for determining whether extubation should be attempted, realizing that all decisions regarding weaning and extubation must be entered into with the understanding that a small and unpredictable percentage of patients will fail extubation. To minimize the risks to these patients, caregivers must make mental and physical preparations to provide an airway and to assist ventilation for all patients prior to extubation.

Table 3. Practical Guidelines for Extubation

No immediate need for mechanical ventilation or intubation	Patient's medical course does not suggest impending respiratory failure or other indication for mechanical ventilation; procedures requiring intubation and general anesthesia are not immediately planned.
Adequate oxygenation and ventilation can be achieved with spontaneous ventilation	Patient's F_IO_2 requirement can be achieved by mask or nasal cannula; patient no longer requires mechanical ventilatory assistance. Quantitative measures—consensus weaning parameters.
Minimal risk for upper airway obstruction	Patient with minimal edema or mass encroachment of the oropharynx and upper airway; oral and upper airway anatomy otherwise normal. Quantitative measures—positive "cuff-leak" test.
Adequate airway protection and minimal risk for aspiration	Patient's level of consciousness and neuromuscular function allow for gag reflex and adequate cough; gastric contents minimized by discontinuation of tube feedings for 4-6 hours prior to extubation. Quantitative measures—none; positive gag reflex helpful.
Adequate clearance of pulmonary secretions	Patient's level of consciousness and muscular strength allow for effective cough; tolerable volume and thickness of secretions that is not worsening. Quantitative measures—none.

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Respiratory Factors in Weaning from Mechanical Ventilatory Support

Neil R MacIntyre MD

I. Introduction & Definitions
II. How Many Patients Need Weaning?
III. Respiratory & Other Factors Involved in Ventilator Dependency Requiring Weaning
IV. Modes of Partial Support & Their Interactions with Ventilatory Muscles
V. Respiratory System Loads & the Weaning Process
VI. Summary

Introduction & Definitions

Weaning is the process of gradually reducing mechanical ventilatory support as the patient's own respiratory system recovers from disease.[1] The term weaning has also been used to refer to the withdrawal of oxygenation support (F_{IO_2} and PEEP) as the patient's gas exchange capabilities improve. For our purposes here, however, weaning refers to the renewal of ventilatory support. Weaning is not synonymous with extubation. Rather, weaning is used in the patient who cannot be totally removed from mechanical ventilatory support but yet is capable of doing some of the work of breathing.

Weaning from mechanical ventilatory support involves using modes of partial support.[2] The advantages of partial over total support are: (1) Partial support either applies less than maximal pressure or applies pressure less frequently.[2,3] Risks of lung injury associated with alveolar distention should, therefore, be reduced. (2) Partial support allows the patient to perform some of the work of breathing, thereby potentially forestalling muscle atrophy.[4] For these reasons, partial support and weaning are usually preferable to the maintenance of high-level or near-total support until the patient can be extubated, in the patient with prolonged ventilatory support needs.

How Many Patients Need Weaning?

Not every mechanically ventilated patient needs gradual withdrawal of mechanical ventilatory support. Patients with rapidly resolving medical problems (eg, asthma and drug overdose) and patients awakening from anesthesia clearly need to have the ventilator withdrawn as soon as their acute medical problem or the effects of anesthesia have resolved. Prolonged use of partial support under these circumstances is clearly contraindicated. In the surgical intensive care unit (ICU), therefore, weaning as we have defined it is needed only in a small minority of patients. Even in the medical ICU, weaning is usually required in only the 20-25% of patients who require the mechanical ventilator for more than 72 hours.[1] It should be noted, however, that, although the number of patients needing weaning with partial support is low, these patients are usually on the ventilator for such prolonged periods of time that they may actually consume the majority of ventilator patient-days in an ICU.

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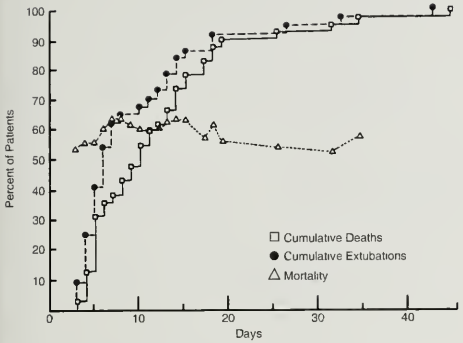

Fig. 1. Outcome of ventilator dependence as a function of days of ventilator dependence in 81 patients requiring ventilatory support for > 72 hours. Note that the likelihood of successful extubation remains constant at 50% to 60% regardless of duration of dependency. (Reprinted from Reference 1, with permission.)

In patients who need weaning, the process is variable in length. In our own medical ICU,[1] weaning periods last anywhere from several days to several weeks (Fig. 1).

Respiratory & Other Factors Involved in Ventilator Dependency Requiring Weaning

A number of reasons for ventilator dependence exist.[5] Nonrespiratory factors include cardiovascular dysfunction, psychological dysfunction, nutritional dysfunction, and multiorgan failure. However, respiratory dysfunction is probably the most important reason for patients' remaining dependent on ventilators. The respiratory reasons can be grouped according to lung 'pacemaker' function (ie, central nervous system respiratory drive), gas exchange function (ventilation-perfusion matching and oxygenation), and mechanical factors (increased impedance demands and increased ventilation demands with respect to respiratory muscle capabilities). The frequency and the success rate of ultimate weaning in patients with these factors in our medical ICU population[1] are given in Tables 1 and 2.

Table 1. Physiologic Determinants of Ventilator Dependence > 72 Hours in 81 Patients*

Determinant	Alone	In Combination with Other Factors	Total
Neurologic factors	8	17	25
Oxygenation factors	8	28	36
Mechanical overload	24	37	61

*Data from Reference 1, with permission.

Table 2. Relationship of Ventilator Withdrawal Success to Initial Determinants of Ventilatory Dependence*

Determinant	Successful (Total Patients)	Success Rate (%)
Neurologic factors		
Alone	7/8	87
In combination	6/17	35
Oxygenation factors		
Alone	4/8	50
In combination	5/28	18
Mechanical overload		
Alone	13/24	54
In combination	11/37	30

*Data from Reference 1, with permission.

Because mechanical factors are among the most prevalent, further discussion of these are in order. Mechanical factors come from a variety of sources (Fig. 2). These include stiff lungs with reduced compliance, narrow airways with increased resistance, and increased ventilation demand (either from increased metabolic demands or increased dead space). There can also be imposed loads from demand-trigger function, demand-flow function, endotracheal tube size, and the inspiratory threshold (due to auto-PEEP).

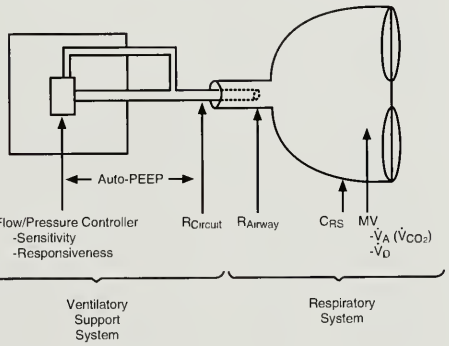

Fig. 2. Loads on respiratory muscles can come either from the respiratory system or from the support system (imposed loads). R = resistance; C_{RS} = compliance of respiratory system; MV = minute ventilation; $\dot{V}_A$ = minute alveolar ventilation; $\dot{V}_D$ = minute dead-space ventilation, $\dot{V}_{CO_2}$ = minute CO_2 production.

Taken together these factors load the muscles during ventilation.[6] Ventilatory failure develops when the load overwhelms respiratory muscle capabilities and produces fatigue (Fig. 3).[7,8] The best measurements of load with respect to muscle function in ventilated patients are the pressure-time product (PTP: the integral of inspiratory pressure over time) and the pressure-time index (PTI: mean in-

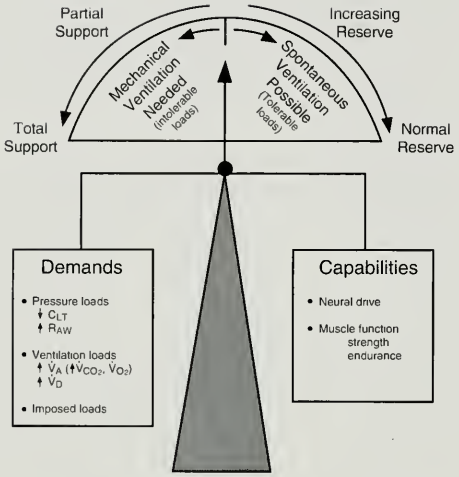

Fig. 3. Ventilatory failure and the need for ventilatory support depend upon the balance between ventilatory muscle demands (loads) and ventilatory muscle capabilities. C_{LT} = lung-thorax compliance, R_{AW} = airways resistance, $\dot{V}_A$ minute alveolar ventilation, and $\dot{V}_D$ = minute dead-space ventilation.

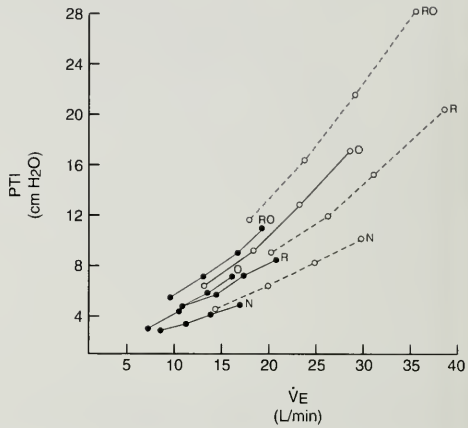

Fig. 4. Lung model data depicting relationship of the pressure-time index (PTI) to muscle loads at a t_I/t_{tot} of 1:3. Loads are represented by both ventilation demands ($\dot{V}_E$ [minute ventilation], horizontal axis) and lung impedances. N = normal resistance and compliance (C_L); R = restricted lung with a C_L of 20 mL/cm H_2O; O = obstructed lung with a doubling of airway resistance; and RO = combined restriction and obstruction. Solid lines represent dead space of 25% of tidal volume, dashed lines represent dead space of 65% of tidal volume. (Reprinted from Reference 6, with permission.)

spiratory pressure × inspiratory time as a fraction of total time, or $P \times t_I/t_{tot}$).[7,8] With data from a lung model, Figure 4 illustrates how differing compliance, resistance, ventilation demand, and dead space affect this measurement of PTI.[6] Whether these pressure demands create fatigue, however, depends upon respiratory muscle function. It appears that under high impedance conditions respiratory muscles can tolerate a PTI of 15% of their maximum pressure-generating capability, as reflected by maximum inspiratory pressure, or MIP.[7] For example, a patient with a MIP capability of 20 cm H_2O could tolerate a mean inspiratory pressure load of 9 cm H_2O with a t_I/t_{tot} of 1:3, or 0.33 (ie, PTI = 0.33 × 9 = 3 cm H_2O), whereas a patient with more normal MIP capability of 100 cm of H_2O could tolerate 45 cm H_2O (ie, PTI = 0.33 × 45 = 15 cm H_2O) at this t_I/t_{tot} without fatiguing. These numbers serve as an illustration of how important the relationship between respiratory muscle loads and muscle function is in determining whether ventilatory failure will occur. Weaning can only proceed as fast as these relationships normalize.

Modes of Partial Support & Their Interactions with Ventilatory Muscles

Approaches to providing partial ventilatory support and thus reloading ventilatory muscles[1,9] include alternating controlled mechanical breaths with periods of spontaneous breathing, alternating intermittent control breaths with intermittent spontaneous breaths (IMV), and using pressure support as partial assistance to every breath. There are similarities among these modes: (1) All require a stable respiratory drive. (2) All require adequate oxygenation because respiratory muscles utilize oxygen. (3) All are designed to gradually increase the respiratory muscle loads as tolerated. Thus, all three should be monitored in the same fashion for patient load-tolerance. In this regard, measurements of respiratory muscle loads (pressure-time products or work of breathing) can be helpful. However, the patient's respiratory pattern is usually the easiest and most reliable guide to patient load-tolerance.[10,11] This means that weaning usually can progress as fast as patient respiratory rate and subjective tolerance permit.

Some differences exist among these modes. Unsupported spontaneous breaths are characterized by a high-pressure, low-volume configuration that may be uncomfortable to patients and, theoretically, are less energy efficient.[9] These characteristics may put the muscles at a disadvantage during this form of ventilatory support as opposed to during pressure support where the partial assistance of every breath reconfigures the work into a more normal pressure-volume relationship.[9] Pressure-limited breaths also tend to be more

Table 3. Weaning Approaches—Gradual Reloading

Characteristic	ACV-T-piece	IMV	PSV
Load quantity	Set by duration of T-tube breathing (guide by load tolerance)	Set by number of mandatory breaths (guide by load tolerance)	Set by level of inspiratory pressure-assist (guide by load tolerance)
Load P-V characteristics	Fixed high P-V by respiratory system and endotracheal tube	Fixed high P-V by respiratory system and endotracheal tube	P-V more normal depending on level of inspiratory pressure assist
Synchrony	Volume-assist has clinician set $\dot{V}_I$, V_T	Volume-assist has clinician set $\dot{V}_I$, V_T	Inspiratory pressure-assist gives patient input on $\dot{V}_I$, V_T
Minute ventilation guarantee	All or none	Minimum guaranteed by mandatory rate	None guaranteed. Minute ventilation depends on patient drive and respiratory system impedances

*ACV = assist/control ventilation; IMV = intermittent mandatory ventilation; PSV = pressure-support ventilation; P-V = pressure-volume; $\dot{V}_I$ = inspired minute volume; V_T = tidal volume.

comfortable in that they can synchronize with patient-ventilator demands.[9] For these reasons, pressure-limited breaths have become a popular form of weaning, and at least one study suggests that pressure-limited ventilation has beneficial effects on the weaning process.[12] Table 3 summarizes the different features of these partial support modes.

Imposed loads can affect any of these partial support modes. These loads come from demand triggers that are insensitive to patient demand, demand flow that is unresponsive to patient needs, endotracheal resistance during spontaneous unassisted breaths, and the development of a threshold load from intrinsic or auto-PEEP in patients with severe air flow obstruction. Each of these sources has been shown to as much as double the work of breathing produced by the respiratory disease itself. Regardless of the weaning mode chosen, careful attention should be paid to minimizing the loads associated with these factors as I discuss later.

Respiratory System Loads & the Weaning Process

It is important to remember that ventilators are supportive, not therapeutic. Managing disease-imposed loads, therefore, requires other specific therapies, such as antibiotics for infections, diuretics for edema, steroids for specific inflammatory states, and bronchodilators for airflow obstruction. The role of the ventilator is to provide adequate support and yet not harm the patient.[2] Plateau pressures, therefore, should be maintained at safe levels (eg, < 35 cm H_2O), F_{IO_2} should be maintained in a safe range (eg, < 0.5-0.6), and the level of support should be such that the respiratory muscles are not fatigued.[2] This is particularly true at night because adequate support for reasonable sleep is important in weaning.

Imposed loads can be minimized in a number of ways. New demand sensors that involve both pressure- and flow-triggering are improvements over the systems of a decade ago.[13] Pressure-limited breaths with their rapid initial and subsequent adjustable flows have been shown to improve patient-ventilator synchrony during assisted and supported breathing and, thereby, to improve comfort.[14] The most recent addition is the ability to adjust the rate of rise of the pressure breath, and this has been shown in selected patients to further enhance synchrony.[15] In the future, systems that can use either distal endotracheal tube pressure or even pleural pressure (as reflected by esophageal pressure) to control pressure during pressure-limited breaths should further enhance synchrony and further minimize patient ventilator dyssynchrony.[16] In addition to distal endotracheal tube sensors, simply using an endotracheal tube of larger diameter can reduce the imposed load during spontaneous breaths. Finally, in the specific situation of an inspiratory threshold load from intrinsic PEEP during airway collapse, small amounts of extrinsic PEEP can serve to equilibrate this pressure in the ventilator circuit and thereby ease the triggering load.[17] One must be careful in

applying PEEP under these circumstances because excessive amounts can further overdistend already overinflated regions of the lung. An esophageal pressure tracing can be particularly helpful in determining an appropriate level of PEEP to use for these circumstances.

Summary

Weaning, or the gradual reduction in partial ventilatory support, is required in only the minority of patients receiving mechanical ventilatory support (those who need prolonged ventilation support, generally more than 72 hours). Weaning techniques involve muscle reloading and generally require intact ventilatory drive, reasonable oxygenation drive, and reasonable oxygenation before they can be initiated. Weaning is best monitored by assessing load-tolerance, which is best accomplished by assessing the ventilatory pattern. Reloading techniques either involve allowing spontaneous unassisted breaths (as T-piece trials or as breaths interspersed with intermittent volume-assisted breaths) or providing partial support of every spontaneous effort through a delivered inspiratory pressure-assist. Different effects on load characteristics and ventilator synchrony can be seen with different modes. Although these effects may affect outcome, disease resolution is generally the rate-limiting step in weaning and the reason why only 50% to 60% of ventilator-dependent patients who need weaning are ever successfully withdrawn from the ventilator.

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What You Need To Know about the Ventilator in Weaning

Catherine SH Sassoon MD and C Kees Mahutte MD PhD

I. Introduction
II. The Role of Ventilator Triggering in Weaning
  A. Pressure-Triggering
  B. Base Flow in Pressure-Triggered Systems
  C. Flow-Triggering
III. The Post-Trigger Period
IV. Measures To Improve Flow Delivery with PSV and SIMV
V. In Summary

Introduction

Weaning is a process leading to discontinuation of mechanical ventilation that consists of slowly decreasing the amount of ventilatory support provided, with the patient gradually assuming a greater proportion of ventilation. Many weaning techniques make use of features incorporated into commercial ventilators: continuous positive airway pressure (CPAP), synchronized intermittent mandatory ventilation (SIMV), airway pressure-release ventilation (APRV), pressure-support ventilation (PSV), volume-support ventilation (VSV), and mandatory minute ventilation (MMV). All of these, except CPAP, are capable of providing gradually decreasing levels of machine support; but it is unclear whether any one is superior to another for ensuring weaning success. However, a recent (1994) multicenter study suggests that weaning with pressure support ventilation (PSV) is more successful than weaning with SIMV or T-piece.[1] With PSV, the patient has some control over the inspiratory flowrate, respiratory rate, and inspiratory duration, and, therefore, patient-ventilator synchrony is better achieved with PSV. The study of Brochard and colleagues[1] suggests that weaning modalities that allow better patient-ventilator synchrony are preferable. Ventilator designs that improve patient-ventilator synchrony and reduce the imposed work of breathing (WOB) have been developed recently. In this paper, we review basic ventilator designs related to patient-ventilator synchrony involving ventilator triggering and the post-trigger period (ie, from onset to end of inspiratory flow) and their impact on WOB. Unfortunately, data on patient WOB remain limited.

The Role of Ventilator Triggering in Weaning

Irrespective of the mode of ventilation employed during weaning, each breath is a patient-initiated breath. Patient-ventilator dyssynchrony occurs when the ventilator is unresponsive to patient effort.[2] Flow-triggering was designed to minimize this problem. Several microprocessor-based ventilators are equipped with both pressure- and flow-triggering. We have previously discussed this topic extensively[3,4] and therefore now review it only briefly and emphasize related topics that have been reported recently. For the ventilator to deliver a flow of gas, the patient must generate a predetermined pressure change with pressure-triggering or a predetermined flow with flow-triggering. The magnitude of the required pressure decline, from the CPAP or positive end-expiratory pressure (PEEP) level, and the time delay are variables that affect patient WOB. Time delay is the time elapsing from the onset of patient effort until the ventilator delivers a flow of gas. Pressure decline should be as small and time delay as short as possible.

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Pressure-Triggering

With pressure-triggering, the patient initiates a breath while both the inhalation and exhalation valves are closed. In the absence of airflow, the area under the pressure and time delay curve (ie, the product of pressure and time) provide an estimate of the isometric work performed by the inspiratory muscles or the patient effort required to open the flow-metering valve. This patient effort is quantitated as the pressure-time product (PTP, an integral of pressure over t_I [$\int Pdt$], with units of cm $H_2O \cdot s$). The rate of pressure decline is affected by the patient's ventilatory drive and inspiratory muscle strength. Both a decreased drive and weak inspiratory muscles reduce the rate of pressure decline, and consequently prolong the time delay. Time delay is obviously a function of ventilator sensitivity.[3] The less sensitive the setting (ie, the greater the pressure change required), the longer the time delay. We have previously described other factors that affect time delay.[4] These factors include errors due to (1) the speed of propagation of the pressure signal, (2) digital sampling of the pressure transducer, (3) the pressure transducing circuit,(4) discrepancies between set and actual PEEP, (5) noise in the circuit, and (6) the flow-metering-valve response time. The pressure sensing site also influences time delay.

During triggering, a change in pressure as a result of patient effort is sensed by the pressure transducer located at the patient-Y or within the ventilator on the inspiratory or expiratory side of the circuit (depending on the ventilator design). The magnitude of the pressure decline is minimally affected by the sensing site because during triggering both the inhalation and exhalation valves are closed, and, therefore, the pressure decline is equal at any site within the ventilator circuit. However, the location of the pressure-sensing site affects the time delay as can be seen by the following illustration—

When the location of the pressure-sensing transducer is on the expiratory or inspiratory side within the ventilator but the pressure-recording site is at the patient-Y, a 12-ms time delay occurs due to the speed of propagation of the pressure signal.

Speed of sound = 0.3 m/ms, or 300 m/s.
Distance between sensing site and pressure transducer = 1.8 m, or 60 in.
Distance between sensing site and flow-metering valve = 1.8 m.
Therefore,

$$\text{time delay} = \frac{2 \times 1.8 \text{ m}}{0.3 \text{ m/ms}} = 12 \text{ ms.}$$

When the pressure-sensing transducer is at the patient-Y, then,

$$\text{time delay} = \frac{1.8 \text{ m}}{0.3/\text{ms}} = 6 \text{ ms.}$$

Similarly, when the pressure-sensing site is located at the trachea, the time delay is 6 ms. However, any delay due to the location of the pressure-sensing site is small relative to the total time delay. Sensing pressure at the trachea has been reported to impose minimal work and probably is more logical than sensing at the patient-Y or at any location within the ventilator.[5] In a mechanical lung model, Banner and co-workers[5] showed that the imposed WOB was markedly reduced when pressure was sensed at the trachea compared to being sensed within the circuit. It should be noted that with both conditions, the pressure-sensing transducer is located on the expiratory side within the ventilator. The difference in imposed work is related to the resistance to gas flow through the endotracheal tube. The pressure-sensing site becomes more important in the post-trigger phase as we discuss later. Unfortunately, under the usual clinical conditions, locating the pressure-sensing site in the trachea is impractical because the site is prone to blockage by secretions and vulnerable to other mechanical abuse related to tracheal suctioning, both of which may prevent accurate sensing of patient effort.

In general, with respect to the imposed work and time delay during ventilator triggering, the pressure-triggering designs of microprocessor-based ventilators have been markedly improved compared to the older generation of ventilators.[6,7]

Base Flow in Pressure-Triggered Systems

A base, or bias, flow has been incorporated into some ventilators with pressure-triggering, and it has been suggested that this base flow, ranging from > 0 to 30 L/min in different ventilators, is useful for minimizing patient effort in opening the flow-metering valve.[8] For the patient to trigger the ventilator in the presence of base flow, the base flow must first decrease and the exhalation valve close to allow the pressure within the circuit to decline to the set pressure-trigger threshold. Depending on the amount of base flow, this may create a PEEP effect and theoretically may increase the patient effort required to open the flow-metering valve. With PSV applied to a mechanical lung model at different levels of pressure support, Konyukov and co-workers[8] recently demonstrated the effect of varying base flow on time delay and work required to trigger the ventilator. With pressure-triggering, work during triggering (trigger work) cannot be calculated because airflow is absent. However, the design of the lung model used in the Konyukov study allows one to measure trigger work from volume and pressure displacements of the "diaphragm bellows" driving the "lung bellows." For a given level of pressure support, time delay and trigger work increase proportionally with increasing base flow (Fig. 1). Thus, base flow with pressure-triggering does not appear

to reduce triggering work, and in fact, may be harmful. The impact of base flow with pressure-triggering on WOB has not been systematically evaluated in patients.

Flow-Triggering

With flow-triggering, both flow sensitivity and the base flow are the set variables, unless the base flow is a fixed value or is automatically set by the ventilator. In some ventilators, the base flow is a fixed value (eg, 2 L/min in the Siemens 300, Siemens-Elema, Solna, Sweden; 10 L/min in the Bird 8400STi; Bird Corporation, Palm Springs CA), or automatically set to twice the flow sensitivity (eg, Hamilton Veolar, Hamilton Medical, Reno NV).[9] Two methods are currently available to flow-trigger the ventilator. In the first method, as the patient inhales, the expiratory flow transducer detects a diminution in the base flow (eg, Puritan-Bennett 7200ae, Puritan-Bennett Corporation, Carlsbad CA; Siemens 300), and in the second method the flow transducer at the patient-Y detects an increase in flow equal to that of the set flow sensitivity (eg, Hamilton Veolar, VIP Bird). Because it is unprotected from secretions and water condensation, a pressure- or flow-sensing device located at the patient-Y requires meticulous care if the device is to remain sensitive. Depending on its magnitude, the base flow may also induce a slight PEEP effect similar to base flow in pressure-triggering, We have previously shown that in healthy subjects, the base flow has no significant effect on the WOB.[10] However, preliminary observations in patients with acute respiratory failure suggest that the WOB with flow-triggered CPAP with a base flow set at 20 L/min is greater than at 10 L/min.[11]

The imposed WOB with flow-triggering is less than with pressure-triggering. However, the differences are primarily related to post-trigger events rather than triggering per se. With 0 cm H_2O CPAP set on the Puritan-Bennett 7200ae and Siemens 300 and each ventilator applied to a lung model, we measured the PTP during ventilator triggering (PTP_{trig}). The flow and pressure sensitivity were set at values commonly used in clinical practice (2 L/min and −1 cm H_2O, respectively). The magnitude of pressure decline and time delay are less than those with pressure-triggering.[3,4] Consequently, PTP_{trig} is also less with flow-trig-

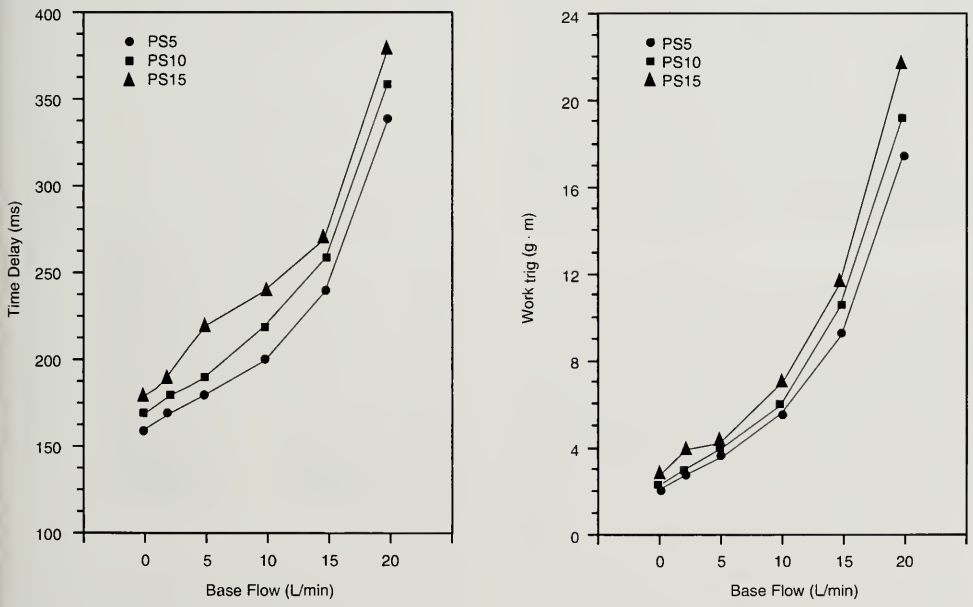

Fig. 1. Effects of varying base flow on ventilator-triggering time delay and trigger work ($work_{trig}$) at various pressure-triggered pressure-support levels. Time delay and $work_{trig}$ increase proportionally with increasing base flow incorporated in a pressure-triggering system. Plotted from data in Table 2 of Reference 13.

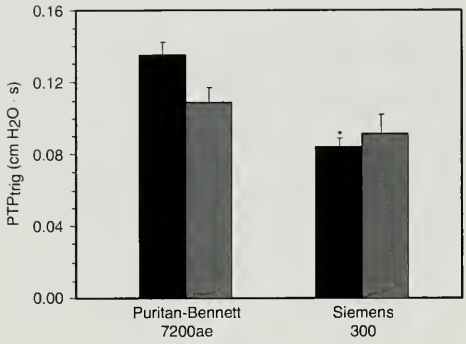

Fig. 2. Pressure-time product during ventilator triggering (PTP_{trig}) measured in a single-compartment mechanical lung model (TTL model 2600i, Michigan Instruments Inc, Grand Rapids MI). The lung model consists of 2 compartments connected with a lifting bar, the 'respiratory muscle' and 'lung' compartments. The respiratory-muscle compartment is driven by a Puritan-Bennett 7200a ventilator. The lung model was set at a tidal volume of 500 mL with rapid ascending and descending flowrates of 45 L/min, compliance of 0.020 L/cm H_2O, and frequency of 12 breaths/min. A 7.5-mm-ID endotracheal tube and an alinear resistor (2.7 cm H_2O at a flow of 1 L/s) connect the lung compartment to the test ventilators (ie, Puritan-Bennett 7200ae and Siemens 300). A pressure-triggered CPAP of 0 cm H_2O was set on the ventilators, with sensitivity set at −1 cm H_2O. With flow-triggered CPAP on the 7200ae, the sensitivity was set at 2 L/min with a base flow of 5 L/min, whereas with the 300, the sensitivity was set at 2 L/min with a fixed base flow of 2 L/min. PTP_{trig} was calculated from the area subtended by the pressure in the lung compartment over time delay. Black bars denote pressure-triggering and gray bars flow-triggering. Values are the average of 3 breaths ± SD. * $p < 0.05$ for pressure-triggered Siemens 300 compared with Puritan-Bennett 7200ae (one-way analysis of variance with Bonferroni's post hoc test).

gering, but as shown in Figure 2, the differences are small. Further, the results obtained in a lung model may not be applicable to patients. During weaning from mechanical ventilation using constant flow and constant pressure SIMV, Giuliani and co-workers[12] compared the effect of pressure- and flow-triggering on PTP_{trig}. Irrespective of the mode of ventilation, PTP_{trig} was significantly lower with flow- than with pressure-triggering during both spontaneous and mandatory breaths (Fig. 3). The investigators also demonstrated that the patient effort required to overcome intrinsic PEEP was also reduced with flow-triggering. With regard to the work imposed during ventilator triggering, flow-triggering provides beneficial effects compared with pressure-triggering. This is because with flow-triggering (1) the patient initiates a breath with both the inhalation and exhalation valves partially opened, (2) the earliest demand for flow is satisfied by the base flow while the flow necessary to meet the flow-sensitivity threshold is being generated, and (3) the trigger threshold is based on flow rather than pressure generation.

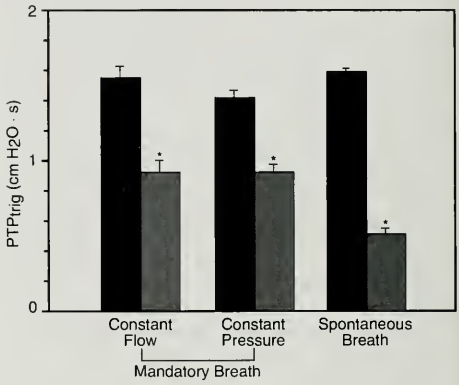

Fig. 3. Esophageal pressure-time product during ventilatory triggering (PTP_{trig}) of the mandatory and spontaneous breaths with constant flow and constant pressure SIMV. Black bars denote pressure-triggering and gray bars flow-triggering. Values are mean ± SE. * $p < 0.01$ flow-triggering compared with pressure-triggering. Plotted from data in Table 3 of Reference 12.

The Post-Trigger Period

Once triggering has occurred, most ventilators are capable of delivering flow ≥ 180 L/min. The amount of flow is determined by a feedback signal sent into the ventilator flow-pressure control algorithm, which depends on the pressure gradients within the ventilator circuit and a target pressure as determined by the manufacturer. The greater the feedback signal, the greater the flow delivered to the patient. The feedback pressure signal can be increased in two ways: (1) by increasing the target pressure above the CPAP/PEEP level and (2) by decreasing the pressure within the circuit. For spontaneous breathing with pressure-triggered CPAP on the Puritan-Bennett 7200ae, the target pressure is 0.5 cm H_2O below the CPAP/PEEP level, whereas with flow-triggered CPAP the target pressure is 0.5 to 1.0 cm H_2O above the CPAP/PEEP level. In the Siemens 300, this target pressure is 2 to 3 cm H_2O above the CPAP/PEEP level for both pressure- and flow-triggered CPAP. A diagram of the pressure gradient between circuit and target pressures is shown in Figure 4. With flow-triggered CPAP of the Puritan-Bennett 7200ae, this pressure gradient is larger than that of the pressure-triggered CPAP. In the Siemens 300, the feedback pressure

Fig. 4. Diagram of pressure gradient (vertical arrows) between circuit and target pressures (shaded bar line) as feedback pressure signal to the ventilator flow-pressure control algorithm of pressure-triggered (PT) and flow-triggered (FT) CPAP of the Puritan-Bennett 7200ae and Siemens 300. The CPAP level is 0 cm H_2O. The pressure-triggered CPAP 7200ae has the smallest pressure gradient, followed by 7200ae flow-triggered CPAP and both pressure- and flow-triggered CPAP of the Siemens 300. The largest pressure gradient is obtained when pressure is sensed at the trachea.

signals to the ventilator flow-pressure control algorithm are equal for both flow- and pressure-triggered CPAP (Fig. 4). As might be predicted, the largest post-triggering PTP is with pressure-triggered CPAP of the Puritan-Bennett 7200ae (Fig. 5). Flow delivery can also be augmented by decreasing pressure within the circuit. When pressure is sensed in the trachea, a large pressure gradient between pressure in the trachea and target pressure is developed

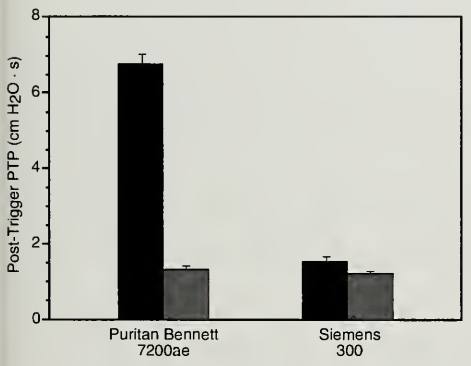

Fig. 5. Pressure-time product during post-triggering measured in a lung model (see Figure 2 for detailed description). Black bars denote pressure-triggering and gray bars flow-triggering. $p < 0.05$ for flow-triggered compared with pressure-triggered CPAP of Puritan-Bennett 7200ae; and pressure-triggered CPAP of Siemens 300 compared with that of the 7200ae.

(Fig. 4). As the feedback pressure signal increases, flow delivery is augmented, which results in decreased work.[5]

With PSV, this target pressure is the level of pressure support. For this reason, the application of small amounts of pressure support (ie, 5 cm H_2O) to pressure-triggered CPAP of the Puritan-Bennett 7200e is adequate to decrease the work of breathing similar to that with flow-triggered CPAP.[13,14] Despite the apparent advantages of flow-triggering over pressure-triggering, other studies[15,16] have failed to demonstrate the beneficial effect of flow-triggering on WOB. Jager and co-workers[15] studied patients with COPD who were recovering from acute respiratory failure. Pressure- and flow-triggered CPAP (Puritan-Bennett 7200ae) were applied in random order for 15 minutes each, with 20 minutes of assist/control ("rest period") between the two CPAP episodes. With flow-triggered CPAP, the base flow was set at 20 L/min and flow sensitivity at 3 L/min. The set trigger sensitivity with pressure-triggered CPAP was not mentioned. In this study, it might be difficult to discern differences in the WOB due to the two CPAP-triggering systems because information on pressure-triggered CPAP is incomplete. Extremely sensitive pressure-triggering (ie, 0.5 cm H_2O) might have been used or the high base flow used with flow-triggering might have increased the WOB as suggested by Mancebo and co-workers.[11] In the study of Nishimura and co-workers,[16] flow- and pressure-triggered CPAP were applied in the rabbits (to simulate pediatric patients) using endotracheal tubes with 3- and 4-mm internal diameters. Pleural pressure swings were less with flow-triggered CPAP only when the animal had a 3-mm endotracheal tube. The advantage of flow- over pressure-triggering is likely to be more pronounced in patients with high ventilatory demand (Figs. 6 & 7[4]), but unfortunately clinical studies are lacking.

Measures To Improve Flow with PSV and SIMV

PSV and SIMV are frequently used for weaning. In this section, we discuss ventilator designs employed for pressure-supported spontaneous breaths (PSV) and mandatory breaths (SIMV) to improve the ventilator's response to demand for flow early in inspiration during the post-trigger period. With PSV, an adjustable initial flowrate that rapidly reaches the optimal pressure-support level is important to decrease the patient's WOB and to improve patient-ventilator synchrony.[17] Optimal pressure support, also called the maximal pressure-support level, is defined as the pressure support that results in an assisted tidal volume of 10-12 mL/kg.[17,18] When a high flow is provided for patients with a high ventilatory demand, it is associated with a large tidal volume, low breathing frequency, and prolonged inspiratory time (t_I), whereas a reduced flowrate re-

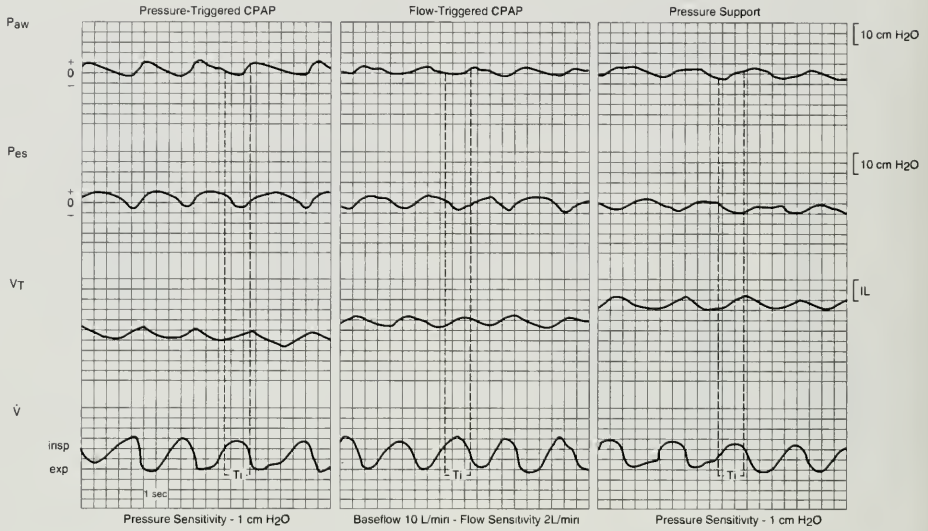

Fig. 6. Airway pressure (P_{aw}), esophageal pressure (P_{es}), tidal volume (V_T), and flow ($\dot{V}$) tracings of a patient without a high ventilatory demand on pressure triggered, flow-triggered flow-by CPAP, and 5 cm H_2O pressure support at a CPAP level of 0 cm H_2O (Reprinted from Reference 4).

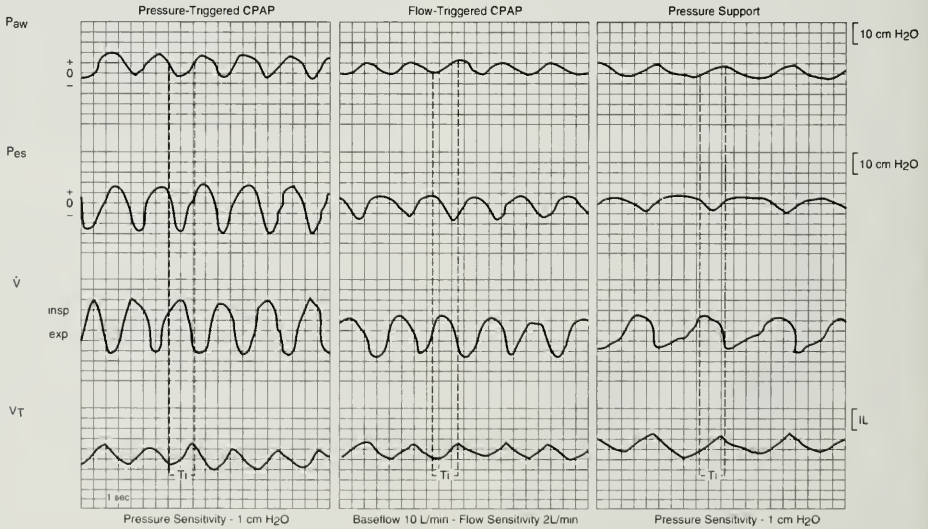

Fig. 7 Airway pressure (P_{aw}), esophageal pressure (P_{es}), flow ($\dot{V}$), and tidal volume (V_T), tracings of a patient with a high ventilatory demand on demand-flow, flow-by CPAP, and 5 cm H_2O pressure support at a CPAP level of 0 cm H_2O (Reprinted from Reference 4).

sults in early termination of pressure-supported breaths, small tidal volumes, increased breathing frequency, and short t_I.[17] In the Siemens 300, initial flow delivery can also be adjusted according to patient ventilatory demand (ie, adjustable inspiratory rise time) when CPAP is used. With CPAP, the impact of adjustable inspiratory rise time on patient WOB or patient-ventilator synchrony has not been systematically evaluated.

With constant-flow SIMV, patient ventilatory demand during the mandatory breaths can be met by either pressure or flow augmentation. This feature is available only on the Bear 1000 (Bear Medical Systems, Riverside CA). The advantage of constant-flow ventilation is the guaranteed flow and tidal volume irrespective of changing respiratory system impedance. However, if a patient's ventilatory demand increases, increasing the set flowrate may result in a concomitant increase in peak-airway pressure. By incorporating pressure augmentation that functions similar to PSV, flow is increased and tidal-volume guaranteed but with a limited increase in pressure (Fig. 8). Preset variables are volume, peak flowrate, and pressure-support level. Pressure augmentation remains active as long as the patient's generated flow is higher than the set peak flowrate. The breath is similar to a pressure-supported breath. On the other hand, constant flow becomes active when tidal volume falls below its set value. In which case, the breath is similar to a constant-flow, volume-cycled ventilator. Amato and co-workers[19] were the first to apply pressure-augmentation to constant-flow, volume-cycled ventilation. This feature can also be applied to the mandatory breaths of SIMV. The authors compared the effect of constant-flow, volume-cycled ventilation with and without pressure augmentation on WOB, PTP, and occlusion pressure after 0.1 second of inspiration ($P_{0.1}$). For pressure augmentation to function, two sources of gas flow in parallel are employed. One source delivers a constant flow with a flowrate as high as 120 L/min, and the other, without any limit in flow delivery maintains a preset pressure-support level. With pressure augmentation, WOB decreased 46%, PTP 52%, and $P_{0.1}$ 43%, and patients with high WOB with constant-flow, volume-cycled ventilation received the greatest benefit when pressure augmentation was added. As an extension to pressure augmentation described by Amato and co-workers,[19] MacIntyre and co-workers[20] described a similar ventilator design combining pressure-limited and volume-cycled breaths that may be used for both mandatory pressure-limited and pressure-supported spontaneous breaths. It also incorporates the adjustable flowrate settings as seen with PSV. In addition to pressure augmentation, the Bear 1000 ventilator is also equipped with a flow-augmentation feature. With flow augmentation (Fig. 9), flow is increased to prevent inspiratory pressure from falling below the PEEP level. Hence, with both flow and pressure augmentation, flow increases with increased patient ventilatory demand, but with flow augmentation, pressure is maintained at PEEP level, whereas with pressure augmentation, pressure increases above PEEP to the set pressure support .[21]

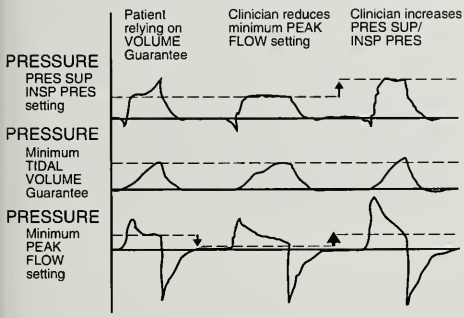

Fig. 8. Pressure, volume, and flow waveforms with pressure augmentation. A set tidal volume is delivered despite decreased patient ventilatory demand and changing respiratory system impedance. With an increase in patient ventilatory demand, the pressure and flow waveforms appear similar to those of pressure-support ventilation. (Reprinted from Reference 21, with permission.)

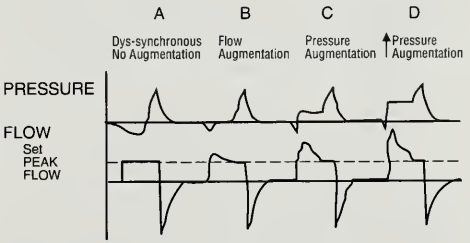

Fig. 9 Pressure and flow waveforms with flow and pressure augmentation. With flow augmentation, when patient effort increases, pressure is increased and maintained at PEEP level. With pressure augmentation, when patient effort increases, pressure is increased to the set pressure-support level.

In Summary

During weaning, flow-triggering is more advantageous than pressure-triggering because of the reduced magnitude of pressure decline and shorter time delay. In the post-trigger period several options are available to augment ventilator flow delivery: (1) with CPAP, the ventilator target

pressure may be increased above the CPAP level or pressure may be sensed in the trachea; (2) with PSV, an adjustable inspiratory flowrate may be provided; (3) with SIMV, pressure or flow may be augmented. It is important to note that despite increased sophistication in ventilator design, little or no evidence suggests that any ventilator feature is superior to simple T-piece weaning in regard to weaning outcome, duration of weaning, ICU or hospital length of stay.

Editor's note: A multicenter study (Esteban A, Frutos F, Tobin MJ, Alia UL, Solsona JF, Valderdu I, et al. A comparison of four methods of weaning patients from mechanical ventilation. N Engl J Med 1995;332(6)345-50.) published since the completion of this paper suggests that T-piece trials lead to earlier weaning than does weaning by IMV or PSV. The abstract of that study is printed on Page 206 of this issue.

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The Effects of Artificial Airways on Airflow and Ventilatory Mechanics: Basic Concepts and Clinical Relevance

Sam R Sharar MD

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  B. Turbulent Flow
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IV. Clinical Relevance of Artificial Airways to Weaning
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Introduction

Placement of an artificial airway (oral/nasal endotracheal or tracheostomy tube) provides a secure and patent upper airway and a point of connection of the lungs to a mechanical ventilator. However, placement of an artificial airway dramatically alters the geometry of the upper airway, changing resistance (R) to airflow, work of breathing (WOB), dead space volume (V_D), and the potential for airway obstruction—all of which can affect both spontaneous and assisted ventilation. These changes in respiratory mechanics are governed by basic principles of fluid mechanics and vary with the type of artificial airway used. The clinician's ability to apply these basic principles to ventilation through artificial airways is essential to maximize the efficiency of assisted ventilation and to predict those patients who can be weaned and extubated successfully.

Fluid Mechanics of Artificial Airways

The primary mechanical effect of bypassing the natural upper airway with an endotracheal tube (ETT) is a nearly three-fold increase in R. Consequently, for the spontaneously breathing patient, WOB must also increase to maintain the same minute ventilation ($\dot{V}_E$).[1] To appreciate the general effects of ETT size and geometry on R, two conditions of airflow—laminar and turbulent—need to be considered. Laminar flow can be described with the simple, linear mathematical relationship of flow ($\dot{V}$) to driving pressure (P)—ie, for every step change in $\dot{V}$, a step change in P (ΔP) results and the size of that interval (step) is constant over all values of $\dot{V}$. This relationship is often used to explain airflow changes seen clinically. However, the reality is that laminar flow occurs only rarely—flow through artificial airways is almost always turbulent. Thus, our simple explanations of airflow in artificial airways based on the laminar-flow model are approximations, at best,

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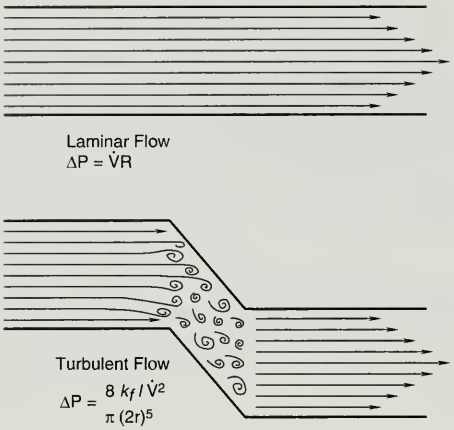

Fig. 1. Schematic illustrating laminar and turbulent flow. Angling causes the laminar flow stream to collide with the tube wall resulting in the transition from laminar to turbulent flow. ΔP = pressure drop, R = resistance, $\dot{V}$ = flow, k_f = gas density friction factor and r = radius. *(Drawing by Steve Bowden)*

and must be interpreted accordingly because, when flow is turbulent, its relationship to driving pressure is not linear. With turbulent flow, the ΔP in response to a step change in $\dot{V}$ is predictable but not constant—and requires a less intuitive and more complex mathematical description. The importance of basic tube characteristics and fluid density on the mechanics of flow through artificial airways is emphasized in the discussion that follows.

Laminar Flow

Laminar fluid flow through a straight tube generally occurs at low flowrates when the fluid molecules stream in a single direction parallel to each other, and eddy or multidirectional currents are absent (Fig. 1). Under such conditions, the relationship between $\dot{V}$, ΔP, and R is linear.

$$\Delta P = \dot{V}R .$$

R is due to friction that is generated among individual fluid molecules and between the outer margin of the moving fluid column and the stationary tube wall. This increased marginal friction relative to the more central zones of the tube results in a parabolic velocity profile where central fluid molecules move faster than those in the boundary layer adjacent to the tube wall (Fig. 1).[2] Once such a velocity profile is achieved, R across the tube can be defined by Poiseuille's law:

$$R = \frac{8l\mu}{\pi r^4},$$

where l is the length of the tube, μ is the fluid viscosity, and r is the tube radius.

Thus, R is proportional to fluid viscosity and tube length, but inversely proportional to the fourth power of r. The result is that R (hence, the ΔP needed to maintain a given $\dot{V}$) increases exponentially with decreases in tube radius. Therefore, when flow is laminar, tube radius is the primary determinant of R.

Turbulent Flow

As flowrates increase or when fluid flow is directed through either a curved tube or a tube with rough internal surfaces, flow is said to be turbulent. This results from flow separation as fluid molecules leave the parallel flow profile seen with laminar flow and disintegrate into more random flow patterns (eg, eddies Fig. 1).[2] Because the random currents are not oriented in the main direction of flow, and may even orient in the direction opposite to main flow, the pressure needed to achieve a given $\dot{V}$ is greater than would be required for the same $\dot{V}$ under laminar-flow conditions. Once turbulent flow conditions have developed, ΔP across the length of such a tube can be defined by:

$$\Delta P = \frac{8\, k_f\, l\, \dot{V}^2}{\pi\,(2r)^5},$$

where k_f is a friction factor dependent on fluid density and irregularities on the internal surface of the tube.

Thus, under turbulent flow conditions, two observations can be made. First, the relationship between the ΔP and $\dot{V}$ is not linear. Second, R is proportional to fluid density, internal-wall roughness, and tube length but inversely proportional to fifth power of its diameter. The result is that R is even more dependent on tube diameter when flow is turbulent than when it is laminar. Furthermore, in contrast to R during laminar flow, during turbulent flow, R is also dependent on fluid density and surface irregularities of the internal tube wall.

The Effect of Artificial Airways on Airflow & Ventilation

Based on the generalized principles of fluid flow through straight tubes, clearly, certain characteristics of

artificial airways including length, diameter, internal surface irregularities (ie, secretions), and the radius of curvature can affect respiratory mechanics.

Length & Diameter

Airway size has an important effect on R and, therefore, on maximum flowrates and WOB. Because R increases linearly with tube length, only slight differences in R can be found between nasal and oral ETTs, and the R of a tracheostomy tube is somewhat less than either of these. However, the strongest determinant of R in terms of tube size is its internal diameter (ID). Merely bypassing the natural airway with even a large-caliber artificial airway results in a significant decrease in airway diameter because the cross-sectional area of an 8-mm-ID ETT is only 50 mm^2 compared to the tracheal and oropharyngeal cross-sectional areas of approximately 150-300 mm^2 and 600 mm^2, respectively. As ID is further decreased, both ΔP and R increase exponentially (Fig. 2). This increase is magnified and is most clinically relevant at flowrates > 1 L/s.[3] This exponential relationship among ID, $\dot{V}$, and R persists across the entire range of ETT sizes, even to the smallest of pediatric

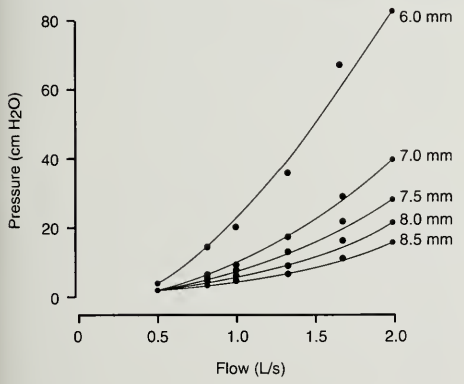

Fig. 2. The pressure drop across endotracheal tubes under constant airflow conditions in vitro increases exponentially with decreases in tube diameter. (Reprinted with permission from Reference 3.)

ETTs.[4,5] For the spontaneously breathing patient to maintain a given minute ventilation ($\dot{V}_E$), increased R must be overcome by increasing ΔP across the airway—a maneuver that may be costly in terms of WOB.[6]

WOB increases exponentially in adults breathing through increasingly smaller diameter ETTs (Fig. 3).[6] Critical influences on WOB have been observed during ventilation via ETTs < 7-mm ID.[6] In healthy adults, data

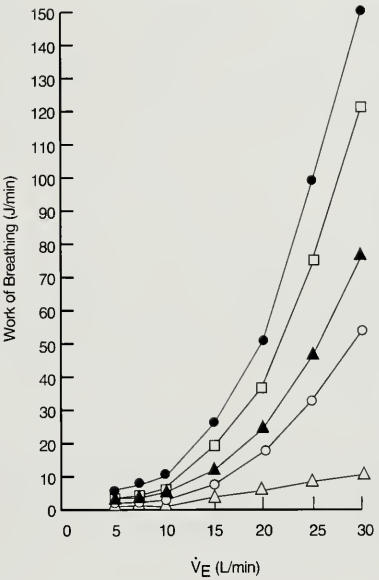

Fig. 3. Work of breathing in healthy, spontaneously breathing adults increases exponentially with decreases in endotracheal tube diameter. ●—● 6 mm, □—□ 7 mm, ▲—▲ 8 mm, ○—○ 9 mm, △—△ 10 mm. (Reprinted from Reference 6, with permission.)

suggest that when $\dot{V}_E$ is 8 L/min or less, little clinical difference in WOB is found among ETTs 7-mm-9-mm ID.[7] However, at higher $\dot{V}_E$s, this increased WOB can become clinically important.[6,7]

Although the respiratory mechanics of spontaneous ventilation through ETTs of various sizes impacts efforts to wean patients from the mechanical ventilator, the effect of tube size on the choice of settings for positive-pressure ventilation has also been studied. In one study of anesthetized, paralyzed, and orally intubated healthy adults, $\dot{V}_E$ as high as 13 L/min could be achieved with ETTs ranging from 8-mm ID down to 5-mm ID, although ΔP across the tube (measured by intratracheal catheter) rose to as high as 42 cm H_2O for ventilation through the smallest tube compared to 8 cm H_2O for the largest tube (Fig. 4).[8] The practical consequence of these findings is two-fold. First, for large $\dot{V}_E$s, maximum ventilator pressures required to ventilate through small tubes can be quite high, and when $\dot{V}_E$ is also high (> 20 L/min), some older ventilators may not be able to achieve high enough driving pressures, necessitating the placement of a larger diameter endotracheal tube.[9] Second, ΔP across small ETTs is so large that for a given $\dot{V}_E$, a change to a larger diameter ETT may reduce peak airway

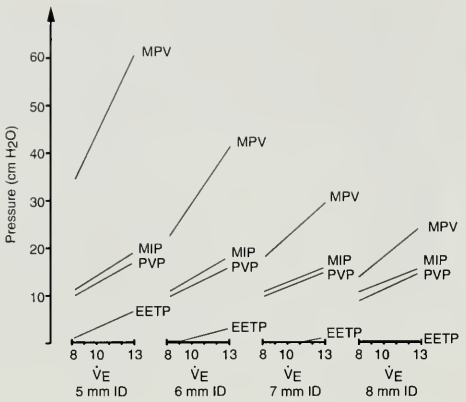

Fig. 4. Ventilator and airway pressures during positive-pressure ventilation of healthy, anesthetized, paralyzed adults as a function of endotracheal tube internal diameter and minute ventilation ($\dot{V}_E$). MPV, maximum ventilator pressure; MIP, maximum intratracheal pressure; PVP, pause ventilator pressure; EETP, end-expiratory tracheal pressure. (Reprinted from Reference 8, with permission.)

pressure measured in the external circuit, yet have little or no effect on peak intratracheal pressure and, therefore, be unlikely to affect the risk of airway and lung injury.

Internal Surface Irregularities

The accumulation of secretions on the inner wall of an artificial airway has two consequences in terms of airflow mechanics: (1) a reduction in luminal diameter and (2) increased internal-wall roughness. Both increase R and WOB. The accumulation of inspissated secretions can be particularly threatening in children due to the risk of airway obstruction that accompanies small ETTs (Fig. 5[10]). Airway obstruction need not be complete to constitute a life-threatening emergency in infants and children.

The Radius of Curvature

The descriptions given earlier for laminar and turbulent flow pertain to flow through straight tubes. They do not adequately describe flow through curved tubes as necessarily occurs in both natural and artificial airways. As laminar fluid flow enters a curved tube, the faster moving central fluid column is directed into the outside wall, producing turbulent flow with multidirectional currents and increasing R. If the rate of flow is to be maintained through this curved tube, ΔP must increase. It can be shown that ΔP across a curved tube is inversely proportional to the radius of curvature of the tube.[11] The radius of curvature is simply the radius of a circle that would be formed if the arc—the curved portion of the tube—were continued until the ends met. Thus, for any given tube size and flowrate, a sharply curved tube (eg, tracheostomy tube), one with a short radius of curvature, should produce a larger pressure drop (hence, increase R) than a more slowly bending tube (eg, oral endotracheal tube) that has a larger radius of curvature.[11]

Fig. 5. Cross-section of an endotracheal tube showing almost complete obstruction of the lumen due to inspissated secretions. The tube was removed from a child after a respiratory arrest following a prolonged general anesthetic for burn excision and grafting. (Reprinted from Reference 10, with permission.)

Because the upper-respiratory tract is not a straight cylinder, all artificial airways curve somewhat in vivo due to the effects of warming and chronic deformation. As stated previously, ΔP and R are inversely proportional to the radius of curvature. Therefore, tracheostomy tubes, with a short radius of curvature (sharply curving) bend at a 90° angle, contribute more resistance than do nasal ETTs that also have a 90° bend but have a longer radius of curvature (gradually curving). Oral ETTs (long radius, > 90° bend) contribute the least angular resistance. This component of R is not easily estimated because of the effects of head and neck position and secretions on tube geometry.

Clinical Relevance of Artificial Airways to Weaning

From the preceding discussion it is clear that the primary mechanical effect of bypassing the natural upper airway with an artificial airway—oral or nasal endotracheal tube or tracheostomy tube—is increased R. Although emphasis here has been placed on tube geometry, other factors associated with artificial airways also contribute to increased R, including reflex bronchoconstriction due to tracheal-ir-

ritant-receptor stimulation by the tube and ineffective clearance of secretions. For the patient who is completely dependent on positive-pressure ventilation, the clinical consequence of this overall change in R is an increased demand on the ventilator to increase driving pressure. Although this demand increases as the tube's ID decreases and $\dot{V}_E$ increases, modern ventilator performance is satisfactory in almost all cases. For the patient being weaned from the ventilator, increased R is manifest by an increased WOB over that imposed by the natural upper-airway,[12] and WOB that changes inversely with ETT ID and directly with $\dot{V}_E$.[6,7,12] In those patients with either borderline pulmonary function or poor muscle strength and endurance, ventilator weaning may be limited by this increased WOB.[13] Using both lung models[14] and human testing in vivo,[12,15] appropriate levels of pressure support that compensate for the R imposed by artificial airways have been determined for a variety of tube sizes and $\dot{V}_E$s (Table 1). This application of pressure-support techniques is further supported by evidence that it minimizes diaphragmatic fatigue during weaning.[16]

Table 1. Pressure Support Required To Overcome Endotracheal-Tube Resistance

Endotracheal Tube	Patient's Minute Ventilation (L/min)		
Diameter (mm)	12	16	20
6	11	17	47
7	5	8	20
8	5	5	9

For a given ID, oral and nasal ETTs demonstrate little difference in their effects on respiratory mechanics. Although nasally placed ETTs are usually longer and offer slightly more curve resistance, their primary drawback is the inherent restriction on ETT size imposed by the size of the nares and nasopharynx that needs not be considered when ETTs are orally placed. However, factors such as infection risk (eg, sinusitis), patient comfort, and anticipated duration of intubation primarily dictate the route of tube placement.

Lung model experiments suggest that WOB is reduced with a tracheostomy tube compared to an endotracheal tube.[17] However, comparison of the mechanics of tracheostomy and ETTs shows that despite obvious differences in geometry, their resistances are not very different. In spontaneously breathing animals, WOB through same-sized ETTs and tracheostomy tubes is similar,[18] suggesting that the mechanical benefit of short tracheostomy-tube length is balanced by its small radius of curvature. In addition, V_D imposed by an 8-mm-ID tracheostomy tube (6-8 mL) is only slightly less than that of 8-mm-ID endotracheal tubes (12-15 mL), both of which are markedly less than the V_D of the natural adult airway (70 mL). However, the practical effect of this decrease in V_D is not known, given that combined anatomic and physiologic V_D may exceed 2/3 of V_T in patients recovering from respiratory failure.[19] In practice, therefore, the decision of performing tracheotomy is generally made using criteria other than respiratory mechanics.

In Summary

The primary effect of artificial airways is that of increased resistance to airflow and the most important determinant of resistance is the internal diameter of that airway. High peak-inspiratory pressure (driving pressure) may be required to deliver normal tidal volumes through small-diameter endotracheal tubes and when very large tidal volumes are administered through larger-diameter tubes. The marked increases in resistance that are caused by the presence of artificial airways translate into increased work of breathing. One could reduce this effect by using larger endotracheal tubes or by implementing a pressure-support protocol during weaning. Of these, the best approach is probably pressure support because increasing the endotracheal-tube size beyond 7-mm ID gains little in terms of work of breathing unless minute ventilation is very high.

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Nonrespiratory Aspects of Weaning from Mechanical Ventilation

David J Pierson MD

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III. Acid-Base Factors in Ventilator Weaning
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Introduction

Most clinicians consider weaning from mechanical ventilation to mean discontinuation of ventilatory support, regardless of the means by which this is brought about and the time it takes to accomplish it. Because most patients receiving short-term ventilatory support do not have serious underlying lung disease and can simply be taken off the ventilator and extubated once the reason for ventilatory support has resolved, weaning in the context in which the term is used in biology—that is, the gradual, stepwise withdrawal of a means of support over a period of time[1]—applies only to a relatively small number of patients. However, both the means for determining whether weaning is likely to be successful and the optimal technique by which to pursue the process remain controversial.[2-6]

The following factors strongly affect a given patient's ability to wean from mechanical ventilation:

- the reason for mechanical ventilation,
- the patient's baseline functional status,
- the current functional status of other organs and systems,
- the duration of the patient's critical illness, and
- the duration of mechanical ventilation.

The clinician's approach to ventilator management prior to attempted weaning may also be important, particularly if this has resulted in respiratory alkalosis, fluid overload, oversedation, or ventilatory muscle fatigue. Many clinicians believe that the ventilator mode selected and/or the weaning method employed are major factors, although this remains unproven despite continued investigation.[7]

An important component of any weaning protocol is selection of the appropriate circumstances for the weaning attempt. This means the best time of day, both from the standpoint of the patient's physiology and psychology and also from the standpoint of available personnel. There is little justification for initiating weaning after a period of

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Table 1. Nonrespiratory Factors in Weaning Patients from Mechanical Ventilation

Category	Factor	Mechanism	Clinical Presentation
Cardiac Status	Acute left-ventricular failure	Increased preload due to increased venous return and decreased pulmonary capillary compression as intrathoracic pressure is reduced	Patient fails weaning, often after initially doing well for 30-60 minutes; may develop acute respiratory and/or metabolic acidosis, hypoxemia, hypotension, chest pain, and cardiac dysrhythmias.
Acid-base Status	Acute alkalosis in patient with underlying CO_2 retention	Loss of pre-existing metabolic compensation for hypercapnia; inability to sustain required minute ventilation and work of breathing	Patient with chronic obstructive lung disease or other cause of chronic respiratory acidosis prior to acute insult fails weaning after several days of ventilation to a lower P_{CO_2} than patient's pH-compensated level.
	Respiratory alkalosis	Depression of ventilatory drives by hypocapnia and alkalemia	P_{CO_2} rises and pH falls during weaning attempt; patient is said to fail weaning if some arbitrary change in these values (such as a 10 torr increase in P_{CO_2}) is used as a criterion for failure.
	Metabolic acidosis	Increase in ventilatory demand to compensate with respiratory alkalosis	Patient may be unable to sustain required increase in minute ventilation and work of breathing to maintain a lower P_{CO_2} to compensate for a lower HCO_3^-.
Metabolic Status	Hypophosphatemia and hypomagnesemia	Ventilatory muscle weakness	Patient fails weaning because of rapid shallow breathing, respiratory distress, and acute respiratory acidosis; maximal inspiratory pressure is decreased.
	Hypothyroidism	Decreased ventilatory drive with possible ventilatory muscle weakness	Rare cause of weaning failure that occurs because of acute respiratory acidosis, with or without respiratory distress.
Drugs	Narcotics, sedatives, tranquilizers, and hypnotics	Depression of ventilatory drive	Patient fails weaning because of acute respiratory acidosis in the absence of tachypnea and respiratory distress.
	Neuromuscular blocking agents	Ventilatory muscle weakness; delayed clearance in patient with renal insufficiency	Patient fails weaning because of rapid shallow breathing, respiratory distress, and acute respiratory acidosis; maximal inspiratory pressure is reduced.
		Ventilatory muscle weakness due to acute myopathy, especially in patients who have received high-dose systemic corticosteroids	Same as above; may have elevated muscle enzymes; can last for weeks or months.
	Aminoglycosides	Neuromuscular blockade	Very rare cause of weaning failure that occurs because of rapid shallow breathing, respiratory distress, and acute respiratory acidosis; maximal inspiratory force is reduced.
Nutrition	Overfeeding	Increased CO_2 production, especially with excessive carbohydrate calories	Patient fails weaning because of excessive ventilatory demand (high minute ventilation requirement to keep P_{CO_2} normal); unusual cause of weaning failure unless very large caloric loads are administered.
	Malnutrition	Effects of acute illness; pre-existing nutritional deficiencies	May contribute to ventilatory muscle weakness, decreased ventilatory drive, impaired immunologic function, fluid retention, depression; distinguishing from other factors is difficult.
Psychological Status	Agitation; "psychological ventilator dependence."	Anxiety, fear, delirium, ICU psychosis, or influence of pre-existing personality factors	Patient becomes agitated and panicky during attempt to reduce or discontinue ventilatory support; can be said to cause weaning failure when other factors are absent.
	Lack of motivation	Depression, effects of drugs, organic brain dysfunction, or influence of pre-existing personality factors	Patient refuses to participate in his own care (eg, mobilization, bronchial hygiene, physiologic measurements), flat affect and immobility in bed (given other factors absent).

prolonged ventilatory support during the late evening or night-time hours because at these times the patient is most likely to be fatigued, and because nursing and respiratory care personnel are likely to be present in fewer numbers and have less time to devote to weaning supervision. Weaning should not be attempted during change-of-shift, when these personnel are distracted by taking report, charting, and other responsibilities. Likewise, ongoing cardiopulmonary resuscitation or some other involved procedure in an adjacent bed, the entry into the intensive care unit (ICU) of a large entourage accompanying physicians' rounds, or arrival of members of the patient's family to visit would all constitute inappropriate settings for a weaning attempt.

The literature on ventilator weaning and the bulk of bedside teaching in this regard focus primarily on respiratory aspects. Assessments of the patient's pulmonary mechanics (eg, inspiratory vital capacity and maximum inspiratory pressure),[8] spontaneous breathing pattern,[9] ventilatory demand (the minute ventilation required to maintain a normal arterial P_{CO_2}),[8] and estimations of the efficiency of gas exchange such as the ratio of P_{aO_2} to F_{IO_2}, are appropriately the main focus of most considerations for weaning. However, even if the measures of all these factors are acceptable, the patient may still be unweanable.

This article focuses on nonrespiratory factors the clinician must consider whenever patients fail to wean on an initial attempt or whenever weaning is undertaken after a period of prolonged ventilatory support.[10] Six separate categories of nonrespiratory factors, as summarized in Table 1—cardiac, acid-base, metabolic, pharmacologic, nutritional, and psychological—are presented.

Cardiac Factors in Ventilator Weaning

Patients with compromised cardiac function may develop acute congestive heart failure when spontaneous ventilation is resumed during ventilator weaning due to a sudden increase in left ventricular preload. As mean intrathoracic pressure drops, venous return to the right ventricle increases and the release of alveolar-capillary compression reduces inspiratory volume and alveolar distending pressure. Two additional factors may help to bring on sudden cardiac decompensation: the modest increase in left ventricular afterload when positive pressure ventilation is removed and the relative cardiac ischemia that ensues when spontaneous ventilation is resumed. These are especially important in patients with increased work of breathing, eg, those with severe chronic obstructive pulmonary disease (COPD).

Lemaire and colleagues[11] studied 15 patients with COPD who could not be weaned following an episode of acute ventilatory failure. Left ventricular dysfunction was demonstrated during weaning attempts, with the mean pulmonary-artery wedge pressure increasing from 8 to 25 mm Hg. Nine of the 15 patients were successfully weaned following vigorous diuresis. Recently, other investigators have confirmed these observations.[12]

Acute cardiac failure should be suspected when a patient initially does well during weaning—eg, breathes comfortably on a T-piece or low-level continuous positive airway pressure (CPAP) for 30-60 minutes—and then develops restlessness, tachycardia, rapid shallow breathing, and diaphoresis. Development of some degree of metabolic acidosis is not unusual, but the patient may not manifest chest pain, dysrhythmias, or hemodynamic instability. If a pulmonary artery catheter is in place, a rise in the pulmonary artery wedge pressure can be observed as a weaning trial in such a patient proceeds. This scenario is most likely to occur in an elderly patient with coexisting cardiac and pulmonary disease.

Acid-Base Factors in Ventilator Weaning

Acute Alkalosis in Patients with Chronic Hypercapnia

Perhaps the most common reason that such patients fail to wean following a period of mechanical ventilation is the presence of relative hyperventilation and respiratory alkalosis and its secondary effect on serum bicarbonate. Table 2 illustrates an all-too-frequent sequence by which hyperventilated patients become 'unweanable' because of the clinician's failure to recognize relative hyperventilation. The table depicts changes that might occur in a patient with severe COPD and underlying stable CO_2 retention, as illustrated in the first column. If such a patient experiences an acute respiratory deterioration and develops acute respiratory acidosis leading to mechanical ventilation, the initial ventilator settings may provide excessive ventilation resulting in an arterial P_{CO_2} level considerably lower than that which the patient normally maintains. This creates an alkalosis that may be as life-threatening as the original acidosis.[13]

Table 2. Blood-gas and pH Values Showing the Impact of Ventilator-Induced Respiratory Alkalosis on Weaning

Variable	Baseline	Respiratory Failure	On Ventilator	After 3 Days	Weaning
pH	7.38	7.24	7.56	7.40	7.24
P_{CO_2} (torr)	58	76	40	40	58
HCO_3^- (mEq/L)	34	36	34	24	26

If the ventilator settings remain unchanged for 2-3 days, and if the patient's renal compensatory mechanisms are intact, then bicarbonate will be lost via urinary excretion, re-

turning the arterial pH to the normal value of 7.40, as shown in the fourth column of Table 2. Although the values for arterial pH, P_{CO_2}, and bicarbonate in that column are entirely normal, they are not normal for this particular patient, who is unable to sustain the level of alveolar ventilation required to maintain them without assistance. Accordingly, unless the original state of compensated respiratory acidosis is restored by reducing the ventilator's contribution gradually (over 2-3 days), weaning attempts are likely to fail.

Respiratory Alkalosis

Hypocapnia and, to a lesser extent, alkalemia depress the hypoxic and hypercapnic ventilatory drives to breathe. If patients are managed with controlled ventilation and respiratory alkalosis, they do not trigger the ventilator or attempt to breathe spontaneously and so are 'easy' to manage. This approach to ventilator management, particularly for anesthesia and surgery, has been used for many years. When taken off the ventilator, a patient whose drive has been suppressed by hyperventilation normally hypoventilates or even remains apneic until a normal CO_2 threshold is reached, and then resumes ventilating at an approximately normal P_{CO_2} and pH. For this readjustment to occur, the P_{CO_2} may need to rise by 5-10 torr or more, depending on the starting values of P_{aCO_2} and arterial pH.

Such changes represent normal physiologic adjustment to acute respiratory alkalosis. However, some weaning protocols rely on the magnitude of P_{CO_2} rise to determine the success or failure of the weaning attempt. If a 10 torr increase in P_{CO_2} is considered a weaning failure and if the patient begins the trial with a P_{CO_2} in the low 30s, then weaning may be delayed or prevented unnecessarily, not because the patient is unable to ventilate appropriately, but, in fact, because that is exactly what the patient is attempting to do. In this case, using the magnitude of CO_2 rise as a determinant of weaning failure is counterproductive.

Metabolic Acidosis

The presence of metabolic acidosis as occurs in patients with renal insufficiency or with the use of carbonic anhydrase inhibitors, such as acetazolamide or sulfamylon, has the effect of reducing the protective function of bicarbonate against acidemia if arterial P_{CO_2} should rise. Patients with metabolic acidosis and those with respiratory insufficiency are particularly hard to wean, especially if they have been critically ill for many days. Because hyperventilation to compensate for metabolic acidosis may exceed the maximum sustainable minute ventilation in such patients, the acidosis may cause patients to remain ventilator-dependent who might otherwise have been weaned.

Metabolic Factors in Ventilator Weaning

Hypophosphatemia

Recently, phosphate deficiency has been recognized as a potential reason for respiratory muscle weakness and failure to wean. This is believed to be due to the effects of phosphate depletion on the high-energy function of the mitochondria. Aubier and associates[14] reported on 8 ventilated patients with acute respiratory failure and severe hypophosphatemia who were unable to wean. The mean serum phosphorous level in these patients was 0.55 mmol/L (normal phosphorus, 1.20 mmol/L). Aggressive phosphate repletion was undertaken, raising the mean serum phosphorous level to 1.33 mmol/L. Transdiaphragmatic pressure, generated by a regimented stimulation protocol, rose from a mean value of 10 cm H_2O to a mean of 17 cm H_2O, indicating improved diaphragm contractility. This study points out the potential adverse effects of hypophosphatemia, although the authors did not actually study weaning, and the frequency with which this metabolic defect results in unweanability has not been clarified.

Hypomagnesemia

Magnesium deficiency has also been reported to be associated with muscle weakness,[15] and at least potentially with the inability to wean from mechanical ventilation.[16,17] Malnourished patients and those with chronic alcoholism are particularly at risk for hypomagnesemia. Magnesium can also be lost from the gastrointestinal tract, and hypomagnesemia is sometimes seen in patients with pancreatitis. As serum magnesium level is not usually included in hospital screening panels, it is important to order this test in patients who fail initial attempts at weaning.

Hypothyroidism

Severe hypothyroidism has several metabolic effects that may adversely affect attempts at weaning. Although these include direct impairment of diaphragm function and actual myxedema of these and other muscles, probably the dominant effect is the blunting of ventilatory responses to hypoxia and hypercapnia. Zwillich and associates[18] demonstrated that patients with severe primary myxedema have markedly blunted hypoxic and hypercapnic ventilatory responses. Patients with hypothyroidism due to removal of exogenous thyroid replacement following total thyroid ablation also had blunted hypoxic drives, although to a less profound degree. In both groups, thyroid hormone replacement resulted in significant improvement in these ventilatory control parameters.

The assessment of thyroid function in critically ill patients is complicated. The classic physical signs of myxedema are difficult to elicit in the intensive care unit, and all are sufficiently variable as to be of limited usefulness, although delayed relaxation of the deep tendon reflexes is perhaps the most helpful of these. The total serum-T4 level is not a helpful stand-alone screen for myxedema for patients in the ICU because of the frequent occurrence of the "euthyroid sick" syndrome. Measurement of thyroid stimulating hormone (TSH) levels, which are markedly elevated in primary myxedema, will avoid this confusion.[19,20]

The Effect of Drugs on Ventilator Weaning

A long list of pharmacologic agents used commonly in the ICU can interfere with weaning. Most commonly encountered of these are sedatives, narcotics, tranquilizers, and hypnotics, and all these drugs are capable of depressing the central ventilatory drive. Therefore, it is important that all such agents be withdrawn or, at least, doses minimized before the attempt at weaning begins.

Neuromuscular blocking agents such as pancuronium, vecuronium, and atracurium render patients incapable of somatic muscle activity, and thus cause ventilatory muscle weakness or paralysis. All muscle relaxants and paralytic agents must be allowed to clear completely before any thought is given to ventilator weaning. In the presence of even mild renal insufficiency, clearance can be delayed, sometimes for days, resulting in persistent ventilatory muscle weakness.

Prolonged muscle weakness following the use of these agents during mechanical ventilation is a recently appreciated syndrome that can substantially increase a patient's requirement for ICU care. Initially reported in patients with status asthmaticus who were treated with sedation and paralysis in conjunction with high doses of corticosteroids, myopathy and/or elevation of serum creatine phosphokinase appear also to occur in patients with other diagnoses who receive muscle relaxants during ventilatory support.[21,22]

Aminoglycoside antibiotics have been reported to be associated with neuromuscular paralysis as a potential cause for weaning failure, although this has occurred most often following peritoneal lavage with agents such as polymyxin, and is rarely encountered with the drugs in common use today.

Nutritional Factors in Ventilator Weaning

Hypermetabolism & Weaning

One prominent reason patients are unable to be weaned from mechanical ventilation is the need for a high minute ventilation. There are three basic physiologic reasons why this may occur: hyperventilation, inefficient ventilation, and hypermetabolism.[2] Hyperventilation is defined by a lower than normal arterial P_{CO_2}, reflecting excessive alveolar ventilation in relation to metabolic need. Reasons for hyperventilation include a deranged central drive, as seen in patients with acute closed head injuries and following certain kinds of drug ingestion; the presence of dyspnea, pain, or agitation; and inappropriate ventilator settings, which cause the patient to receive more ventilation than is necessary.

Inefficient ventilation is indicated by an increased dead-space fraction (V_D/V_T). A number of familiar causes may be responsible, including pulmonary thromboembolism, which reduces perfusion to areas of lung that continue to be ventilated; severe obstructive lung disease, such as chronic obstructive pulmonary disease or asthma; and acute hyperinflation as seen with rapid ventilator cycling, shortened expiratory times, and failure to fully exhale between breaths, manifested by auto-PEEP (inadvertent positive end-expiratory pressure). Another cause of inefficient ventilation is the ventilation-perfusion ($\dot{V}_A/\dot{Q}$) imbalance seen in the late phase of the acute respiratory distress syndrome (ARDS), which is dominated by areas of high ventilation and low perfusion, producing a dead-space effect.

Hypermetabolism as a reason for inability to wean reflects an increase in the demand for ventilation rather than necessarily a problem with the ventilatory apparatus. It is indicated by an increased CO_2 production ($\dot{V}_{CO_2}$), which is readily measured by collecting exhaled gas for 3 minutes in a Douglas bag and analyzing this mixed expired gas via capnograph or arterial blood gas machine. Indirect calorimetry using so-called metabolic carts has become popular during recent years for measurement of $\dot{V}_{CO_2}$) and oxygen consumption ($\dot{V}_{O_2}$), but these expensive devices have often proven disappointing in clinical practice, primarily due to the many variables that have to be considered and the need for training and careful adherence to protocols by the personnel operating them.[23] The time-honored (and less expensive) Douglas-bag method circumvents some of these difficulties but not others, and it cannot be used with a number of current ICU ventilators.

Possible reasons for increased $\dot{V}_{CO_2}$ as a reason for high minute ventilation include increased demand—as seen in sepsis, fever, shivering, seizures, or agitation—and excessive caloric intake. Providing a patient with more calories than needed for nutritional support can result in generation of excessive CO_2, which then has to be ventilated away, perhaps exceeding the patient's abilities to do this spontaneously.

The amount of CO_2 generated in the breakdown of different nutritional substrates varies according to the nature of the substrates. This is commonly measured using the respiratory quotient ($RQ = \dot{V}_{CO_2}/\dot{V}_{O_2}$).[24] The usual North

American mixed diet results in an RQ of approximately 0.8; intake of pure fat results in an RQ of 0.7, whereas a carbohydrate diet results in an RQ of 1.0. In addition, if an excessive amount of overall calories is provided, such that lipogenesis occurs in the liver, the RQ of this process can be as high as 8.0, a phenomenon, however, that seldom occurs in critically ill patients.

Malnutrition & Weaning

Focus on the nutritional aspects of critical illness in general, as well as of ventilator weaning, has increased markedly during the last 15 years.[25,26] Although there remains a paucity of solid scientific evidence to back up much of current practice, it seems clear that patients may fail to wean because of too little nutrition, on some occasions because of too much nutrition, and perhaps also because of the wrong kind of nutrition.[27]

Malnutrition has several well-documented effects on the respiratory system, including impaired ventilatory muscle function, diminished ventilatory drive, and a variety of impairments of the immune system.[27] Perhaps most important are the effects on the respiratory muscles. The diaphragm and other muscles of ventilation quickly undergo several adverse processes when the body's caloric and other nutritional needs are not met. These include deterioration of the contractile properties of the diaphragm, such that this vital muscle becomes both weaker and less efficient in its function.

Although it has not been documented specifically in patients who fail to wean from mechanical ventilation, at least two aspects of the normal ventilatory control mechanism have been shown to deteriorate under conditions of semi-starvation: the normal hypoxic ventilatory response and the hypercapnic ventilatory response.[28] In addition, a number of changes in the body's immune system, though not specific determinants of the ability to wean on a given day, contribute to increased susceptibility to infection and impaired infection-fighting ability.

Malnutrition develops or worsens in most patients during acute hospitalization, and this applies especially to those ill enough to require mechanical ventilation. Several studies have concluded that aggressive support of nutrition in patients being weaned from mechanical ventilation can increase the likelihood of success.[29,30] However, shortcomings in the design of these studies illustrate the difficulty of performing good research on critically ill patients, and point out the need for a better database to guide nutritional therapy during weaning.

Bassili and Deitel[29] reviewed the records of 47 patients who were ventilated >3 days. They determined that 93% of those deemed to have received adequate nutritional support were successfully weaned whereas among patients without adequate nutrition, only 55% were successfully weaned. In another study, Larca and Greenbaum[30] reviewed the courses of 14 medical ICU patients who had been 'unweanable' during attempts in that setting. After transfer to another area of the hospital and attempts at intensive nutritional support, 6 of these patients failed to respond and could not be weaned, whereas 8 of them showed objective evidence of response to nutritional support (such as increased serum albumin and transferrin levels) and were eventually weaned. Both of these studies suffer from inability to control the selection of patients, and it is possible that numerous factors in addition to nutritional status affected the ability of the patients to wean. Clearly, a prospective, randomized, controlled trial of intensive nutritional support as an aid to ventilator weaning would be most desirable, but in the years since these two articles were published no such satisfactory study on this topic has appeared.

Several general conclusions can probably be drawn about nutritional support during ventilator weaning, even in the absence of controlled clinical trials. It is clearly important to give *enough* nutrition to avoid or to combat malnutrition, but it is also important not to provide an *excess*, particularly of carbohydrate calories. Whether such nutritional support should be administered by the enteral or parenteral route is debatable, and both routes have practical drawbacks in treating critically ill patients. Many hospitals now have a nutritional support service, which can be extremely helpful in designing an individual patient's nutritional support regimen. When available, this service should be utilized whenever a patient fails initial attempts at weaning that follow a prolonged period of mechanical ventilation.

Psychological Factors in Ventilator Weaning

This last category of nonrespiratory aspects of ventilator weaning is perhaps the most difficult to objectify. Although the usual weaning protocols that have evolved from the studies of Sahn and Lakshminarayan[8] and of Yang and Tobin[9] focus on attempts to remove the subjective from assessments of weanability, subjective rather than objective factors may be particularly important in weaning after prolonged mechanical ventilation, especially in individuals with severe underlying lung disease. In 1985, Muir and associates[31] reported, in abstract form, a study that compared intermittent mandatory ventilation (IMV) and T-piece weaning techniques in patients with COPD following an episode of acute respiratory failure. In their 30 patients, who were randomized to IMV weaning versus T-piece trials, they assessed maximum inspiratory pressure, mouth occlusion pressure (an index of central respiratory drive), forced vital capacity, and abdominal

paradox as means of objectifying success or failure in the weaning attempt. They also examined something they called the "nurse wean index."

The outcomes of the study by Muir et al[31] were ultimate weaning success, the time it took for successful weaning, and an attempt to identify a best predictor of weaning. Of all the measurements, only the nurse wean index could be shown to be distinguishable as an independent "best predictor" of whether weaning would be successful. Although it is not stated in the paper, the "nurse wean index" consisted of simply asking the most experienced nurse in the intensive care unit whether he or she believed a given patient would be able to be weaned, expressing this probability on an analog scale of one to five.

The fact that this index was a better predictor than the usual variables measured to predict weanability is an important (although incidental) finding in this study, and demonstrates the value of an experienced clinician's judgment in cases of difficult weaning after prolonged ventilation. No doubt the judgment of such an individual takes into consideration the patient's overall level of illness, something that Morganroth and associates[32] also found to be an important determinant of weaning after prolonged mechanical ventilation in patients with acute illness.

Such things as the patient's attitude and underlying personality are important as well. When dealing with a patient who has failed initial attempts to wean and has been critically ill for a number of days, it is important to think about intangibles as well as things that may be objectively measured. These intangibles include entities that I refer to as the "slug factor" and the "twinkle factor."

Patients with a high "slug factor" are those who lie passively in bed and resist attempts of caregivers to mobilize them and to facilitate secretion clearance and hesitate to assume increasing responsibility for their own care. Patients with a high "twinkle factor" are those who are optimistic and highly motivated to recover from their illness. Although there are no objective data on these arbitrary descriptors, clearly the higher one's "twinkle factor," and the lower one's "slug factor," the more likely one is to be weaned successfully after an episode of acute respiratory failure.

Anxiety, fear, delirium, and ICU psychosis are common in critically ill patients, particularly when they require many days of ventilatory support and other ICU care.[33,34] Depression is also common in such patients. Clearly, if the likelihood of eventual ventilator weaning is to be high, correct identification and management of these factors is important.

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Dyspnea during Weaning

John J Marini MD

I. **Definitions**
II. **Qualitative & Psychometric Measures of Dyspnea**
III. **Quantifiable Measures of Dyspnea**
   A. **Maximum Static Pressure, Pressure-Time Index, & Maximum Inspiratory Pressure**
   B. **Inspiratory Occlusion Pressure ($P_{0.1}$)**
   C. **Respiratory Muscle Pattern**
   D. **Segmentation of Breathing Pattern**
   E. **Possible Role for Biofeedback**

Definitions

The first task to confront when attempting to discuss dyspnea in patients receiving mechanical ventilation is definition of the term. There are many definitions that can be defended, but for the purposes of this discussion, I will equate dyspnea with breathlessness: an awareness of uncomfortable breathing. Although under normal conditions, we generally remain unaware of our breathing, dyspnea is not the simple perception of effort, and although dyspnea is uncomfortable, it is not painful. Rather, dyspnea is the feeling that "something is wrong" with the act of breathing, a feeling that will eventually prove intolerable. Although there are no physical signs of dyspnea that are both sensitive and reliable, diaphoresis, nasal flaring, vigorous use of accessory muscles, and a facial appearance of fright or desperation are highly suggestive.

Qualitative & Psychometric Measures of Dyspnea

Even though the psychological perception of stimulus strength may closely parallel its actual value, dyspnea does not relate linearly to the intensity of the stimulus that evokes it. Dyspnea can be assessed qualitatively by directly interviewing the patient who is able to communicate effectively. Attempts to *quantitate* dyspnea have been made using a variety of psychometric instruments.[1,2] On the modified Borg scale, dyspnea is scored numerically from 0-10 using descriptors to cue the subject to the appropriate value[3] (Fig. 1). Despite widespread use in research, variants of the Borg scale do not permit fine distinctions among grades of dyspnea severity. An attempt to obviate this problem can be made by using a visual analog scale (VAS).[5,6] With this interviewing instrument, dyspnea is graded on a continuous basis by asking the patient to demarcate where along an unmarked line he currently rates the difficulty of breathing (Fig. 2). The line is anchored by descriptors only at the two extremes of the scale, which ranges from no breathlessness at all to the greatest degree of breathlessness possible. The visual analog score is sim-

Modified Borg Category Scale

0	Nothing at all
0.5	Very, very slight (just noticeable)
1	Very slight
2	Slight
3	Moderate
4	Somewhat severe
5	Severe
6	
7	Very severe
8	
9	Very, very severe (almost maximal)
10	Maximal

Fig. 1. The modified Borg Scale for dyspnea. (From Reference 4, with permission.)

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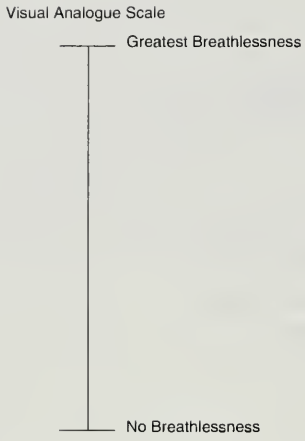

Fig. 2. A simplified visual analog scale for breathlessness. (From Reference 7, with permission.)

ply the distance (in millimeters) from the origin to the demarcated point along the line. Theoretically, the VAS allows an observant patient with reproducible responses to report the level of breathing discomfort without either forcing the score into a crude category or priming the subject with suggestive descriptions of sensation. Although useful for serial comparisons in an individual subject, neither the Borg nor the VAS can be used effectively in comparisons across different patients.

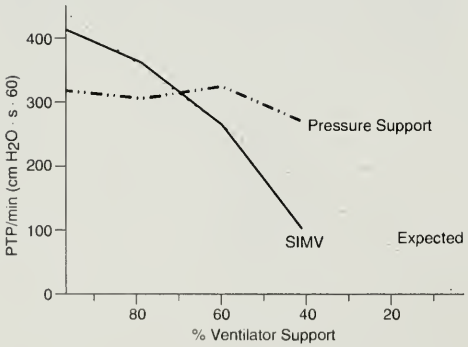

Fig. 3. Differences between SIMV and pressure support relative to the assistance provided by the ventilator per minute as the level of intended support is reduced.

Quantifiable Measures of Dyspnea

Dyspnea is a primary reason why weaning fails; we tend to reconnect the ventilator when the patient appears distressed, even if gas exchange has not deteriorated. Knebel and others in my group[8] have used VAS to score anxiety and dyspnea to demonstrate that patients may respond to the withdrawal of pressure support and SIMV with similar levels of dyspnea and anxiety, even though these two weaning modes tend to reload the respiratory system in different ways[9] (Fig. 3).

Establishing effective communication with patients receiving mechanical ventilation is often impossible. Techniques useful in research (such as the VAS and Borg) do not serve well in the clinical setting and are not feasible for patients who fail to communicate effectively with the caregiver. Therefore, a reliable *involuntary* measure of dyspnea would be valuable. For most subjects, the sense of breathlessness relates to the relationship between the work load present and their ability to perform it. Ideally, we would be able to measure directly the subject's perception of effort. It is currently believed that the intensity of phrenic nerve activity correlates strongly with dyspnea.[1] Lacking such direct measures, physicians often rely on such crude indices as externally measured work, developed pressure, muscle oxygen consumption, or respiratory electromyography.

Maximum Static Pressure, Pressure-Time Index, & Maximum Inspiratory Pressure

Growing evidence suggests that dyspnea can be tracked effectively by measures of respiratory mechanics and workload, provided that those measures are referenced to the capability of the patient. One such measure is the pressure-time index (PTI), which attempts to quantify the inspiratory pressure generated per breath (P) as a fraction of the maximum static pressure (P_{Imax}) that can be generated:

$$PTI = \frac{(P)(t_I)}{(P_{Imax})(t_{tot})},$$

where t_I = inspiratory time and t_{tot} = respiratory cycle time.

Judging from elegant studies performed in the human physiology laboratory, a pressure time index > 0.15 cannot be sustained indefinitely.[10] In work we have previously completed, the PTI for normal subjects stressed with inspired CO_2 or imposed airway resistance bears a convincing relationship to dyspnea scored by the visual analog technique.[11] Low dyspnea scores are recorded until the PTI approximates 0.07-0.08, beyond which scores rise exponentially (Fig. 4). Precise quantitation of the pressure

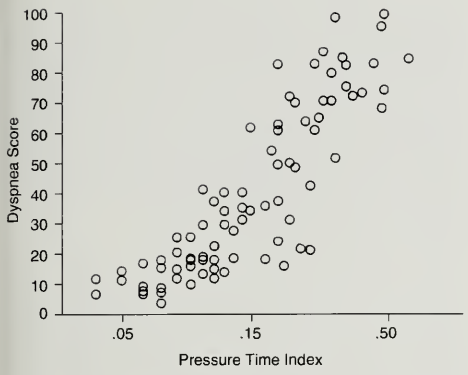

Fig. 4. Correlation between the pressure time index and visual analog dyspnea score in normal subjects. Note that significant changes in dyspnea are not reported until the pressure time index exceeds ~0.07.

terms used in the index often proves difficult in practice. It is quite simple to measure inspiratory time fraction, and the mean or maximum pressure per breath can be estimated from knowledge of minute ventilation, inspiratory and expiratory resistance, and compliance.[12] The maximum pressure inspired against an occluded airway, however, is generally thought to require voluntary effort. Unfortunately, full-intensity effort can seldom be guaranteed in the clinical setting.

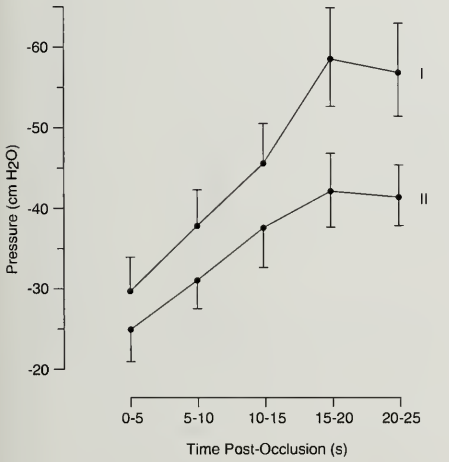

Fig. 5. Estimates of maximal inspiratory pressure with (I) and without (II) use of a one-way (expiratory) valve. (From Reference 13, with permission.)

A number of years ago, our group suggested using a one-way valve to help the patient achieve maximum inspiratory pressure (MIP). In a study comparing methods for measuring the MIP, we found that considerably greater pressures were generated when a one-way valve (directed toward expiration) was used.[13] Approximately 8 breaths or 20 seconds of repetitive breathing efforts were required before a maximal effort could be elicited (Fig. 5). In subsequent work, we asked whether using a one-way valve would allow us to record MIP in a *poorly cooperative* subject.[14] In that study, we found that many cooperative patients improved their MIP value convincingly when coached to do so and that stimulating breathing with use of external-circuit dead space improved the concordance between voluntary and involuntary measures of MIP. A subanalysis indicated that for patients who have at most a moderate ventilatory drive ($P_{0.1}$ < 2 cm H_2O), occlusion with a one-way valve may indeed elicit a near maximal response reliably—even without subject cooperation (Fig. 6).

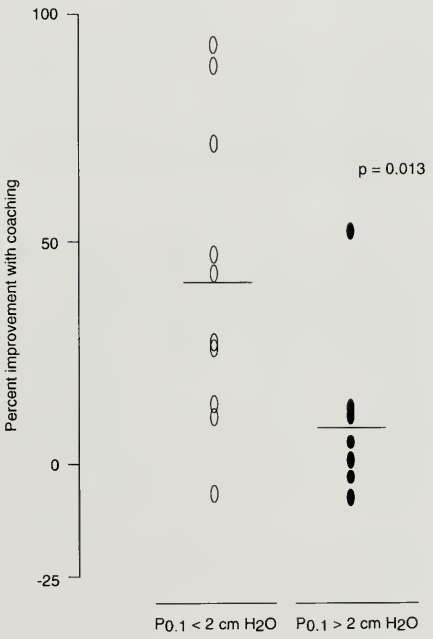

Fig. 6. Difference between voluntary and involuntary estimates of maximal inspiratory pressure as a function of ventilatory drive. (From Reference 14, with permission.)

The proto-inspiratory occlusion pressure, or $P_{0.1}$, provides another indicator of breathing effort and dyspnea. The $P_{0.1}$ is the inspiratory pressure observed 100 milliseconds after surreptitious occlusion of the airway, and is believed to reflect the patient's drive to breathe.[15] Indeed, the correlation of $P_{0.1}$ with Borg scale ratings of stimulus intensity in normal subjects indicates that there is a linear relationship between them, independent of whether breathing is stimulated by increased minute ventilation requirement or increased respiratory loading[1,16] (Fig. 7). Perhaps it is not surprising, therefore, that it has been suggested that $P_{0.1}$ indicates the likelihood of weaning success or failure.

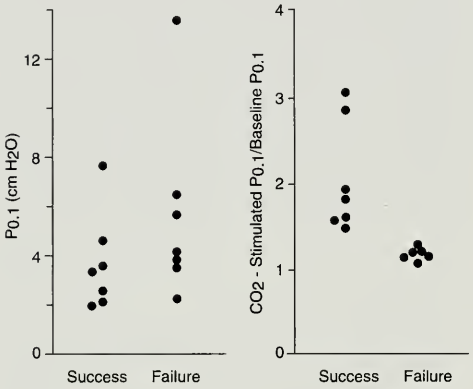

Fig. 7. Comparison of spontaneous and CO_2-stimulated $P_{0.1}$ values for patients who succeed and fail in a weaning attempt. (From Reference 20, with permission)

In studies conducted over the last 12 years by Holle, Sassoon, Murciano and their respective colleagues,[17-19] the $P_{0.1}$ proved to be a reliable index of weaning success; high values (> 6 cm H_2O) predicted failure. At approximately the same time, the CO_2-stimulated $P_{0.1}$ was suggested by Montgomery and colleagues[20] to provide better discrimination between those able and those unable to wean from mechanical ventilation. Montgomery et al reported extensive overlap between weaning success and failure for patients with the same $P_{0.1}$ value (Fig. 8). However, those who generated a higher $P_{0.1}$ in response to a CO_2-stimulated breathing challenge showed adequate power reserves and improved likelihood for successful discontinuation of machine support.

The raw value for $P_{0.1}$ imperfectly indexes ventilatory drive because the occlusion pressure is influenced by muscular strength. However, the $P_{0.1}$ referenced to maximum inspiratory pressure (P_{Imax}) would seem to be a more promising index, in that similar values of $P_{0.1}/P_{Imax}$ correlate with similar Borg-scale ratings of stimulus intensity for both normal subjects and for patients with COPD, across a considerable range of breathing stresses[1,21] (Fig. 9). Fernandez, Benito, and colleagues have used this ratio effectively as a weaning index.[22] Such an index, therefore, may provide a quantifiable physiology-based measure of perceived breathing intensity and dyspnea.

Respiratory Muscle Pattern

The pattern of respiratory muscle activation has been correlated with the extent to which the respiratory muscles are taxed. Such discoordinated patterns as abdominal paradox and respiratory alternans suggest that the patient is approaching the limits of breathing reserve and, by implication, may signal respiratory distress.[23,24] These signs, however, are nonquantifiable, 'all or none' observations. If an impedance plethysmograph is used, the excursions of both the thoracic and abdominal compartments (effective or not) can be summed and averaged, to yield the mean compartmental amplitude. The mean compartmental amplitude divided by tidal volume can help quantify the extent of

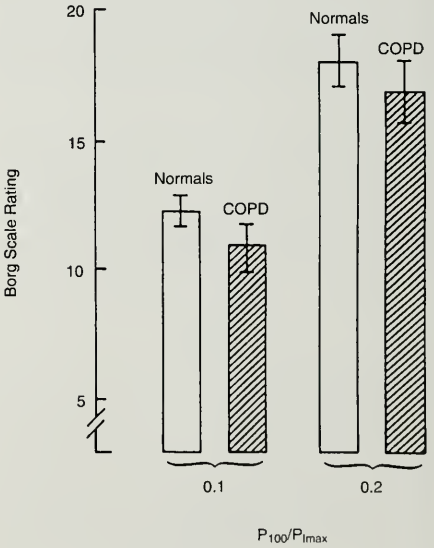

Fig. 8. Correlation of Borg scale rating of dyspnea to $P_{0.1}$ (P100) normalized to maximum inspiratory pressure for normal subjects and patients with COPD. (From Reference 1, with permission.)

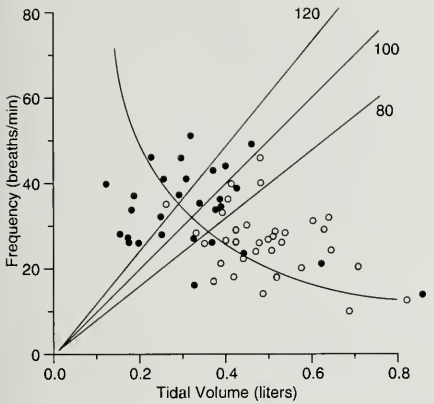

Fig. 9. Relationship of frequency to tidal volume averaged over the first minute of ventilator disconnection. Lines radiating from the origin indicate frequency-to-tidal volume ratios of the indicated magnitude. Successful attempts to discontinue ventilator support (open circles) tended to be associated with ratios < 100; failures are associated with ratios > 100 (closed circles). (From Reference 28, with permission.)

breathing inefficiency and muscle discoordination,[23,25] but unfortunately, this is not an index that is used widely.

Segmentation of the Breathing Pattern

The breathing pattern itself—the way in which minute ventilation is partitioned between tidal volume and frequency—may provide a ready indicator of the stress under which the respiratory muscles operate. During normal exercise, frequency and tidal volume rise together. In the first phase of exercise, tidal volume rises somewhat faster than frequency, but as the stress increases and the anaerobic threshold is crossed, the tidal volume reaches a maximum while frequency continues to rise. A patient nearing the limits of endurance also tends to increase frequency and reduce tidal volume, perhaps because—for the same minute ventilation—small tidal volumes are less energy costly than large ones.

Tobin and colleagues studied the breathing patterns of normal subjects[26] and patients[27,28] undergoing rapid reloading of the respiratory system and quantified how rapidly such changes occurred. From their data, these investigators suggested that analysis of the breathing pattern might be helpful in assessing the degree to which the respiratory system was loaded in relation to its capacity. Data from patients receiving mechanical ventilation who underwent 1-minute disconnection from the machine suggest that the frequency-to-tidal-volume ratio may index the degree to which the respiratory muscles remain compensated. Ratios exceeding 100 breaths · min^{-1} · L^{-1} indicate the strong likelihood of continued ventilator dependence, whereas values less than 100 units suggest that the patient may be weanable (Fig. 9). This simple index may indeed be a useful indicator of the relative intensity of respiratory muscle loading and, therefore, indirectly of dyspnea.

Possible Role for Biofeedback

Finally, it is worth noting that the pattern of ventilation and the $P_{0.1}$ can be modified independently by appropriate biofeedback. As shown in a study by Holliday and Hyers, ventilator-dependent patients can be trained to alter both breathing pattern and $P_{0.1}$ in response to visual cues.[29] These interesting (but largely uncorroborated) data hold out the possibility that we may be able to attenuate dyspnea by voluntary readjustment of the breathing pattern itself.

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Psychological Factors in Weaning from Mechanical Ventilatory Support

Neil R MacIntyre MD

I. Introduction
II. Causes of Psychological Disturbances in the ICU
  A. Disease-Related Factors
  B. ICU Environmental Issues
  C. Care Delivery Issues
III. Manifestations of Psychological Disturbances in the ICU
IV. Incidence of Psychological Disturbances in the ICU
V. Management of Psychological Disturbances
  A. Environmental Issues
  B. Communication Issues
  C. Specific Weaning Plans
  D. Drugs
VI. Clinician-Related Psychological Factors

Introduction

Weaning from mechanical ventilatory support can be a prolonged process in patients with a slowly resolving pulmonary disease. In patients with the acute respiratory distress syndrome (ARDS), the mean weaning time is often 1-2 weeks, whereas in neurologic injury, the process can take months.[1,2] The rate-limiting factor in weaning is usually the rate of resolution of the underlying disease. This is affected by the specific therapies instituted and the adequacy of other support techniques (eg, cardiovascular, nutritional, and renal.). Although patient attitude can have profound effects on the aggressiveness of the weaning process and on the ultimate outcome, psychological factors are less appreciated. In this paper, I review the causes, manifestations and management of psychological factors in weaning.

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Causes of Psychological Disturbances in the Intensive Care Unit

Disease-Related Factors

A number of disease-related issues contribute to psychological disturbances in the intensive care unit (ICU).[3] Severity and duration of the disease are two particularly important factors.[4] The patient's underlying chronic health and age are also contributing factors.[5,6] Specifically, older patients with chronic debilitating diseases usually have concomitant depression and anxiety that accompany disability and dependency on professional health care. Long-term use of such medications as sedatives, catecholamines, steroids, and xanthines may also contribute to psychological dysfunction.[7]

The physiologic effects of the acute disease can contribute to important psychological dysfunction. With regard to lung disease, hypoxemia and acid-base distur-

bances clearly have effects on psychological function;[6] electrolyte and nutritional disturbances associated with ICU care can also contribute. In the lung disease patient, dyspnea is an additional important factor. Dyspnea is caused by a variety of factors that includes hypoxemia, acidosis, and mechanical work load.[3] Other contributing factors that are poorly understood include lung irritant-receptor function that gives the patient increased subjective dyspnea out of proportion to mechanical and gas exchange abnormalities. Complicating this is the fact that psychological factors such as anxiety potentiate the dyspnea response.[2,7] Thus, it should not be surprising that the acutely hypoxemic, acidotic, and muscle-overloaded elderly patient with chronic lung disease, underlying depression, and deranged irritant receptors often experiences severe psychological stress when receiving mechanical ventilation.

ICU Environmental Issues

The ICU environment—an environment characterized by a variety of monotonous noises interrupted by shrill alarms[10]—provides a severe stress to one's psychological well being.[3,9] Severe sensory deprivation in this sterile environment often leaves the patient to stare at a white ceiling, unable to communicate with staff or family. Mobility is usually restricted, and the patient lives essentially in a 'straight jacket.' Finally, depending upon the patient's level of awareness, there is the associated stress of being dependent upon a variety of technologic systems for life itself.

Care Delivery Issues

The delivery of ICU care can also have direct psychological impact.[3,9,11] Foremost among these factors is the disruption of the physiologic sleep pattern.[12] Normal sleep lasts up to 8 hours and is cyclic in nature as the brain proceeds through various stages to rapid-eye-movement (REM) sleep in roughly 90-minute periods. This cycle is often disrupted in the ICU, and the resulting sleep deprivation is thought to be a major factor in the delirium and other features of ICU psychosis.[3,9,12,13]

A variety of ICU procedures can also be psychologically traumatizing. Surgical procedures done with suboptimal anesthesia or sedation can be markedly stressful. Even procedures as simple as vascular access can be painful and stressful if the patient is not provided with adequate analgesia. An ICU procedure unique to the mechanically ventilated patient is endotracheal suctioning. Patients coming out of ICUs have reported this to be a particularly uncomfortable sensation and use terms such as suffocating to describe it.[14,15] Bronchoscopies and alveolar lavage are doubtlessly similar.

A variety of drugs have the potential to impact the patient's psychological status.[16-18] Sedatives, narcotics, paralytics, and barbiturates are often given to help control psychological dysfunction; they may, however, serve to worsen it through paradoxic reactions, cognitive dysfunction, and sleep disturbances. In the respiratory patient, catecholamines, xanthines, and steroids are also used and can have adverse psychological effects.[17] Hypoperfusion and the need for vasoactive drugs may also contribute to psychological dysfunction.

Manifestations of Psychological Disturbances in the ICU

There are a number of manifestations of psychological disturbances in the ICU. Delirium refers to the phenomenon of time and place disorientation.[13] Sleep disturbances and sensory deprivation, coupled with other factors noted are major factors in the production of the delirium. Early in an ICU stay, patients will report hallucinations and terrifying nightmares, and these often precede other manifestations of psychological dysfunction.[3,9,13] Patients can experience both fear of dying and of abandonment, and this results in manifestations of agitation, anxiety, and depression. Cognitive dysfunction has also been reported in a number of COPD patients in the ICU[19] (Fig. 1[20]).

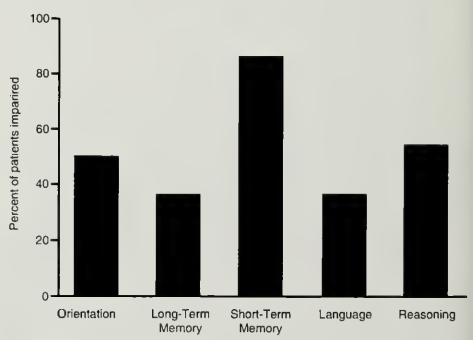

Fig. 1. Presence of cognitive deficits in 28 patients receiving prolonged mechanical ventilation. (Reprinted from Reference 20, with permission.)

During the weaning process these psychological disturbances can be increased for a number of reasons.[2] Patient anxiety may be increased because of increasing dyspnea and, particularly in patients with chronic disease, a fear of death and abandonment as the devices are withdrawn. This anxiety may manifest itself as demanding or questioning behavior. It can also result in apparent patient withdrawal

from the staff. Weaning may also increase depression, particularly in patients with chronic disease who have a long-felt dependence on outside help.[6] This manifests itself as poor motivation, despair, and sometimes even suicidal tendencies. It should be noted that both anxiety and depression may increase secondary-gain behavior on the part of the patient. This should be recognized as a call for help rather than being a source of irritation.

Incidence of Psychological Disturbances in the ICU

As noted, psychological disturbances are clearly related to length of stay and severity. For instance, it has been shown that in patients who remain in a coronary care unit longer than 5 days, the incidence of psychological disturbances is 47%.[21] In patients in an ICU following heart surgery, the incidence of psychological disturbances was 29% in American Heart Association Class I patients (ie, those with mild cardiac dysfunction) but rose to 100% in Class IV patients (ie, those with severe cardiac dysfunction).[4] Underlying chronic disease with associated depression and anxiety also increases the incidence of acute disturbances. Cognitive dysfunction has been found to occur in almost 100% of COPD patients in an ICU.[19]

Management of Psychological Disturbances

Environmental Issues

A number of environmental issues can be addressed to reduce the psychological stress on patients being weaned from mechanical ventilation.[21] Individual rooms reduce extraneous noise. Having these rooms equipped with windows has been shown to reduce by as much as two thirds the incidence of delirium in ICU patients.[13] Nocturnal interventions should be kept to an absolute minimum. No good reason exists for taking routine vital signs or performing other routine procedures during the sleeping hours. Monotonous sounds such as ventilators and pumps should be minimized to the extent possible. For example, some of this equipment can be moved outside the room. Nuisance alarms are a particular source of stress to patients, and every effort should be made to reduce alarms to only those that are truly important and affect decision making.[22] During the day, patient mobility should be maximized. Indeed, moving a patient to a chair or even wheeling the patient out into the hall can greatly improve psychological status. Clocks, calendars, and pictures in the room also help keep the patient oriented to time and place and provide important sensory input. The effects of radio and television have not been carefully studied, but it would seem reasonable that this type of input could be beneficial.

Communication Issues

Patients need to communicate.[21] Clinician eye contact and touching are important communication tools. Giving the intubated patient access to writing equipment or even devices such as talking tracheostomy tubes may be helpful. Above all, clinicians must be patient in trying to communicate with their patients; communication may be a struggle but is of vital importance in keeping the patient psychological stable.

Specific Weaning Plans

A number of weaning maneuvers can minimize psychological stress.[21] This is particularly true in the long-term patient with slowly resolving lung injury. First, a schedule can be helpful in allowing the patient to know when increased work loads are going to be placed on him or her. Second, it is imperative that these work loads not fatigue the patient. Possible reductions in imposed work should be a particular focus (eg, endotracheal tube and valving systems).[1,2] Dyspnea often increases as work loads are increased, and it is important for the clinician to determine whether this increased dyspnea reflects serious physiological disturbances (such as hypoxemia, hypotension, or muscle overload) or a more subjective response related both to irritant receptors and anxiety. The former needs to be treated with increasing levels of support, whereas the latter needs to be managed by reassurance and encouragement. Strategies that are designed to fatigue the diaphragm and then rest it do not make physiologic sense and probably should be avoided. Day-night considerations are particularly important in the long-term weaning patient. Specifically, the patient should be given whatever is necessary to let them rest at night and only have them doing significant work during the day. Ventilatory support (either the IMV rate or the pressure support level) should probably always be increased at night in long-term weaning patients. Third, encouragement and notations of progress can provide important positive feedback to patients. Encouraging self-care as much as possible also helps reduce psychological dependency. Biofeedback using physiologic signals from either lung volumes, breathing pattern, or cardiac signals has been shown to be useful in asthma, but the role of biofeedback in the weaning process remains controversial. Research is needed on this topic.

Weaning strategies that incorporate careful attention to psychological needs can be helpful in getting patients off the ventilator. Units that are specifically designed to deal with the long-term weaning patient often have better success than do acute-care hospitals. This is not due to the use of one particular technique but rather reflects a compre-

hensive approach to weaning that involves both physiologic and psychological considerations[23] (Fig. 2).

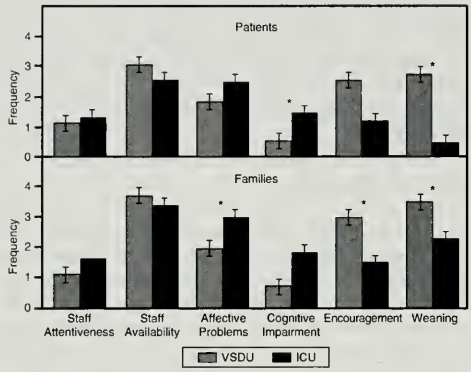

Fig. 2. Mean ratings by 28 patients (upper panel) and families (lower panel) for perceptions of overall experience in the Ventilator Rehabilitation Unit (VSDU) and Intensive Care Unit (ICU) settings. Each Item was rated by the patient or family member on 5-point interval scales according to the perceived extent or frequency of each criterion: 0 = never, 1 = occasionally; 2 = sometimes, 3 = often, 4 = very often (Reprinted from Reference 20, with permission.)

Drugs

Anxiolytic, neuroleptic, and antidepressant medications all have a role in treating anxiety, agitation, and depression in the ICU. Again, caution must be exerted in the use of these drugs because of potential respiratory depression as well as potentiation of other aspects of psychological dysfunction (eg, sleep disturbances).[17,18,24] Opiates are particularly good at blocking dyspnea, especially dyspnea associated with lung edema and irritant dysfunction, and have been used to make patients with chronic dyspnea more comfortable.[25] Their role in facilitating the weaning process, however, is probably limited given the fact that neurological depression and decrease in respiratory drive are associated with them. Aerosolized opiates may be an attractive alternate in this regard.[26] Further research is needed here.

Clinician-Related Psychological Factors

The potential for unnecessary clinician anxiety also exists during the weaning process. Much of this is related to concerns about reducing support too fast in slowly recovering patients. One certainly does not want to prolong the process through the induction of unnecessary fatigue. To address this, sophisticated mechanical prediction models exist and these can be used to guide the weaning protocol. On the other hand, aggressive, frequent reduction of the level of support by skilled practitioners, such as nurses or therapists at the bedside, as rapidly as the patient can subjectively tolerate it may be a more attractive approach than routines done once or twice daily using lung mechanics or blood gas data.[1] Indeed, merely assessing patient comfort, breathing frequency, and breathing regularity may be the most useful guide to weaning, and frequent adjustments by bedside personnel may be the most aggressive way to safely wean patients.[1,2]

Clinician depression can also be a problem if the weaning process seems to be 'dragging on.' It must be remembered, however, that serious lung injury may take weeks to resolve and it may be months before a patient with neurologic dysfunction can safely breathe on his or her own.[1] An example of this is the injured phrenic nerve following coronary bypass grafting, which often takes several months to recover.

There is no doubt that unnecessary connection to a mechanical ventilator increases the risk of lung aspiration, airway injury, and ongoing psychological disturbances. On the other hand, however, premature extubation has its own risks. The clinician must be patient with the protracted nature of the recovery process and at the same time be as aggressive as possible in removing support safely and comfortably for the patient.

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Noninvasive Positive-Pressure Ventilation in Acute Respiratory Failure: Review of Reported Experience with Special Attention to Use during Weaning

Catherine SH Sassoon MD

I. Introduction
II. NPPV in Acute Respiratory Failure
  A. Patient-Ventilator Interface
  B. Modes of Ventilation
  C. Etiology of Acute Ventilatory Failure
  D. Patient-Selection Guidelines
  E. Timing of NPPV Application
  F. Early Predictors of Success
III. NPPV for Patients Who Refuse Intubation
IV. NPPV in Discontinuation from Mechanical Ventilation
V. Considerations for Use of NPPV in Facilitating Weaning

Introduction

The long-term benefit of noninvasive positive-pressure ventilation (NPPV) in patients with chronic ventilatory failure[1-5] has encouraged its use in the management of selected patients with acute ventilatory failure of different etiologies,[6-22] including chronic obstructive pulmonary disease (COPD),[6-19] asthma,[9,14] cystic fibrosis,[8,11] chest-wall disease,[8,9,11,14] obstructive sleep apnea,[10,20] pneumonia,[6,12,14,15] pulmonary fibrosis,[11,15] adult (now, acute) respiratory distress syndrome (ARDS),[6,9,12] congestive heart failure,[6,9,12,14,21] drug abuse,[15] and surgery.[12,15,22] The application of NPPV has been extended to supporting patients in acute respiratory failure who refuse intubation[14,23,24] and to facilitating discontinuation from conventional mechanical ventilation.[25,26] These applications were reviewed in this journal in 1994.[27-29] In this paper, I review the reported experience with NIPPV in acute respiratory failure and give special attention to use during weaning from conventional mechanical ventilation.

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NPPV in Acute Respiratory Failure

NPPV has gained increasing acceptance in the management of acute ventilatory failure because of its advantages over conven-

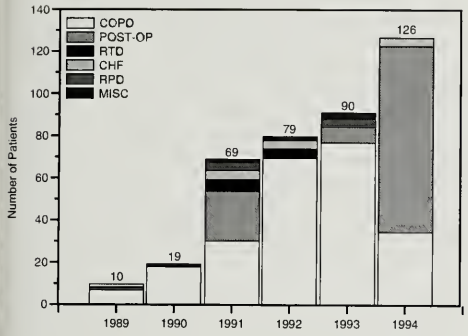

Fig. 1. The number of patients reported to receive noninvasive positive pressure ventilation (NPPV) for treatment of acute ventilatory failure from 1989 through late 1994 (the time of this writing). The number of patients receiving NPPV continued to increase steadily, the majority consisted of patients with chronic obstructive pulmonary disease (COPD) and postsurgical (post-op). RTD = restrictive thoracic disease; RPD = restrictive parenchymal lung disease; CHF = congestive heart failure; MISC = miscellaneous conditions. Number above bars is the total number of patients receiving NPPV.

tional mechanical ventilation, yet it has its measurable limitations (Table 1). To date, the application of NPPV has been reported in a number of studies[6-22] involving nearly 400 patients, with an overall success rate of 70%. Of these patients, 75% have been reported in the last 2 years (1993 & 1994, Fig. 1). It should be noted that the relatively high success rate can be partly attributed to the stringent criteria used for patient selection for admission into the study (Table 2).

Table 1. Advantages and Limitations of Noninvasive Positive-Pressure Ventilation.

Advantages	Limitations
Avoids endotracheal intubation and its related complications	Requires patient cooperation
Preserves airway defense mechanism	Limits airway access and suctioning of secretions.
Improves patient comfort	Causes mask discomfort and feeling of suffocation (face mask)
Preserves speech and swallowing	Causes mask-related complications: facial ulcers due to mask pressure; eye irration, rhinitis, dry nose
Reduces the need for sedation	Causes aerophagia, increased risk of aspiration in the event of vomiting (face mask)
Allows intermittent apptication	Air leak
	Transient hypoxemia from accidental mask removal

Table 2. Patient Selection Criteria for the Application of Noninvasive Positive Pressure Ventilation in Acute Ventilatory Failure*

To participate in this investigation, subjects must

- Have acute ventilatory failure or insufficiency with acute respiratory acidosis and respiratory distress (tachypnea and use of accessory muscles)
- Be cooperative and able to synchronize with the ventilator
- Be able to achieve a proper mask fit
- Have intact bulbar function (intact cough reflex and swallowing function)
- Be hemodynamically stable (no uncontrolled cardiac dyshythmia or hypotension)
- Not have multiorgan dysfunction
- Not have active upper gastrointestinal bleeding
- Not have excessive secretions
- Not have facial trauma

*Adapted from Reference 28

Because the patient-ventilator interface, the mode of ventilation, and the etiology of the ventilatory failure might also influence the success rate of NPPV, we pooled all patients receiving NPPV reported between 1989 to the present[6-22] and compared the success and failure rates. We excluded studies in which noninvasive continuous positive airway pressure (CPAP) was employed or when the goal of the study was primarily for supportive treatment of patients refusing intubation.[23,24] Success rate was defined as the percentage of patients who tolerated the device with improved gas exchange and in whom endotracheal intubation was avoided.[30,31] The selection criteria for patient inclusion in lhose reported studies[6-22] are summarized in Table 3.

Table 3. Advantages and Disadvantages of Nasal- and Face-Mask Interfaces

Nasal Mask	
Advantages	Disadvantages
Freedom with talking, eating and drinking	Mouth leak
Less risk for aspiration in the event of vomiting	Improper fit in edentulous patients
Less risk for aerophagia	
Less risk for claustrophobia	

Face Mask	
Advantages	Disadvantages
No mouth leak	Interferes with talking, eating and drinking
Edentulous state does not interfere with mask fit	Increases risk for aspiration in the event of vomiting
	Increases risk for aerophagia
	Increases risk for claustrophobia

Patients were excluded when any of the following were present: life-threatening arrhythmia,[9] hemodynamic instability,[9,17,22] multiple organ dysfunction,[15,22] unstable angina or recent myocardial infarction,[9,17] need for endotracheal intubation to protect the airway or to manage secretions,[9,17] status asthmaticus,[15] severe disease not attributable to chronic respiratory disease,[16] inability to properly fit the mask, and lack of patient cooperation.[9]

Patient-Ventilator Interface

The application of a nasal or face mask could potentially influence the success rate of NPPV. Each has its advantages and disadvantages as shown in Table 3. The application of a nasal or face mask did not yield any significant difference in the success rate of NPPV, 72% vs 70%, respectively (Fig. 2).

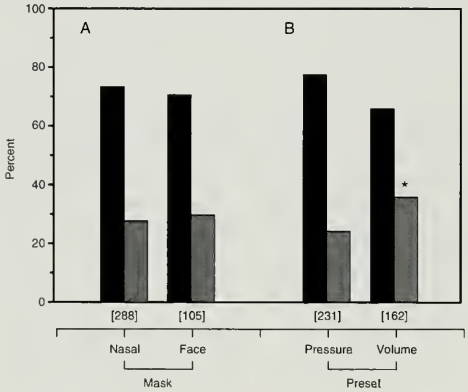

Fig. 2. Graph showing success (black bar) and failure (gray bar) rates (%) of patients receiving noninvasive positive pressure ventilation by nasal or face mask (A) and the use of pressure- or volume-preset ventilation (B). Numbers in brackets indicate the number of patients in each group. * $p < 0.01$ (χ^2) for volume-preset compared with pressure-preset ventilation. Data are from References 6-22.

Modes of Ventilation

In contrast to the patient-ventilator interface, the mode of ventilation employed (ie, pressure-vs volume-preset ventilation, or VPV, appears to influence the success rate. With pressure-preset or pressure-support ventilation (PSV), the success rate of NPPV was 77%, whereas this value was 64% ($p < 0.01$, χ^2) with VPV. These results are in contrast to the randomized prospective study of Vitacca and colleagues[18] who evaluated the efficacy of PSV and VPV in a homogenous population of patients with COPD. In that study 16 patients were randomized to PSV and 13 patients to VPV. Two of the 16 patients treated with PSV and 3 of the 13 patients treated with VPV required endotracheal intubation (a success rate of 87.5% with PSV vs 77% with VPV). Mean duration with both modes of ventilation was similar. Despite similar success rates between PSV and VPV, the authors reported a better patient compliance when PSV was employed.[18] The lack of a significant difference between PSV and VPV might be due to the relatively small number of patients. However, in this pooled patient analysis, the relatively high success rate of postsurgical patients treated with PSV[22] may have favored PSV. In the study of Pennock and co-workers,[22] 88 of 110 postsurgical patients were successfully treated with nasal NPPV using PSV. When those patients were excluded, the difference in the success rate between the two modes of ventilation was no longer significant. I do not find the relatively higher success rate with PSV compared with VPV surprising because with PSV the patient has some control over inspiratory flowrate and respiratory timing.[32] Further, PSV allows better patient-ventilator synchrony and lower peak airway pressure. PSV also better compensates for air leak than VPV, but is less reliable in delivering the target tidal volume in the face of changing respiratory system impedance. A large, prospective, randomized controlled trial with the different patient-ventilator interface and modes of ventilation is still lacking.

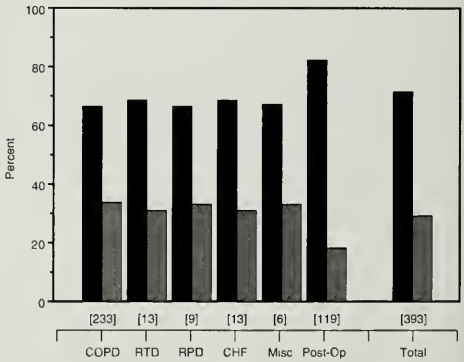

Fig. 3. Graph showing success (black bar) and failure (gray bar) rates (%) of patients receiving noninvasive positive pressure ventilation according to the etiology of the acute ventilatory failure. Numbers in brackets indicate the number of patients in each group. Data are from References 6-22.

Etiology of Acute Ventilatory Failure

The etiology of the ventilatory failure did not appear to influence the success rate of NPPV, although there was a trend toward a higher success rate (82%) in the postsurgical patients[6-22] (Fig. 3). It should be noted that most of the reports involved uncontrolled trials in which the majority of the patients had COPD.

followed by postsurgical patients (Fig. 3). The number of patients in the other disease categories was small. The group with restrictive parenchymal disease (RPD), in which oxygenation is a major problem, consisted of ARDS (n = 3), pneumonia (n = 3), and pulmonary fibrosis (n = 3). The success rate in this group of patients has to be interpreted with caution because impaired oxygenation alone makes success less likely.[15] The miscellaneous disorders consisted of hypothyroidism (n = 1), drug abuse (n = 2), and unknown etiology (n = 3).

In patients with COPD, Bott and colleagues[16] conducted the only prospective, randomized, controlled trial comparing nasal NPPV plus conventional treatment with conventional treatment alone. The selection criteria for entry into the study ensured that all patients had severe COPD with ventilatory failure. Thirty patients were randomized to nasal NPPV plus conventional treatment and 30 patients to conventional treatment alone. Four patients randomized into NPPV did not receive the treatment, and two of them died. Two patients could not cooperate, one patient requested withdrawal of all treatment, and one patient could not breathe through his nose. Nasal NPPV was more effective in correcting the hypercapnia and acidosis and in improving dyspnea than conventional treatment alone. Median hospital stay was similar for both treatment groups. Mortality rate at 30 days was less in the NPPV group than in the conventional treatment group (1/26 vs 9/30). However, when mortality rate was analyzed on an intention-to-treat basis (3/30 vs 9/30), the difference was not statistically significant. Because the patients treated with NPPV fared better, the authors recommend NPPV in all patients admitted with acute exacerbations of COPD and ventilatory failure who do not respond promptly to conventional therapy. Despite the proven efficacy of NPPV for the treatment of acute ventilatory failure in this prospective well-controlled study, guidelines for patient selection, timing for implementation and discontinuation, and early predictors for success remain to be determined.

Patient-Selection Guidelines

Meyer and Hill[31] proposed patient-selection guidelines for NPPV application in acute ventilatory failure (Table 2) based on selection criteria used in previous studies. The patient must meet the criteria for acute ventilatory failure or insufficiency with evidence of acute respiratory acidosis and respiratory distress (ie, tachypnea and/or the use of accessory muscles). Patients presenting with hypoxemia alone are poor candidates for NPPV.[15] In addition to meeting the criteria for acute ventilatory failure, patient cooperation, ability to synchronize with the ventilator, and proper mask fitting are important for the success of NPPV. In the studies in which information on patient tolerance and difficulty in mask fitting are available, 37 of 88 patients (42%) who failed NPPV failed due to intolerance to the equipment or excessive air leak. Also, patients should be hemodynamically stable (defined as the absence of hypotension and/or uncontrolled cardiac dysrhythmias). Patients with poor airway control, excessive secretions, active upper gastrointestinal bleeding, and multiorgan dysfunction are poor candidates for NPPV.

Timing of NPPV Application

The timing for the application and discontinuation of NPPV are unclear. Soo Hoo and co-workers[19] suggested that a delay in the application of NPPV might have influenced their poor results. In 14 episodes of acute ventilatory failure in 12 patients with COPD, NPPV was begun following an unsuccessful trial of conventional therapy. The success rate of NPPV in their study was 50%. On the other hand, in the study by Bott and colleagues[16] in which NPPV was started at the outset in conjunction with conventional therapy, the success rate was significantly higher (87%). However, it should be noted that in the study of Soo Hoo et al,[19] the major cause of failure was excessive air leak and patient inability to synchronize with the ventilator. It appears that timing of NPPV has little influence on the success rate but that the patient-ventilator interaction is of prime importance. More difficult to assess than timing for the initiation of NPPV is timing for its discontinuation. In successful cases, patients require progressively shorter periods of NPPV in subsequent days and can finally be discontinued. But, depending on the severity and chronicity of the underlying pulmonary disease, some patients may continue to receive daytime and/or nocturnal long-term domiciliary NPPV.[14,16]

Early Predictors of Success

Few data are available on early predictors for successful use of NPPV. If such predictors were available, they would be useful for avoiding delay of endotracheal intubation. Mechanisms for the efficacy of NPPV in acute ventilatory failure are primarily related to NPPV's providing increased alveolar ventilation and decreased work of breathing,[7] and in selected patients improved ventilation-perfusion matching.[14,15] A significant reduction in P_{aCO2} and improved P_{aO2} during the course of NPPV were demonstrated in patients who were successfully treated with NPPV and for whom arterial blood gas data were available (Fig. 4). Several investigators proposed that a reduction in P_{aCO2} and/or an increase in pH within the first hour or two are good predictors for successful NPPV.[9,19] In the study by Chevrolet and co-workers[11] of 6 patients (3 COPD; 3 restrictive thoracic/lung disease) treated with NPPV, all showed a significant decrease in P_{aCO2} (from 74 to 49 torr) within an average of 3.2 hours. However, only 3 of the 6 patients were successfully treated with NPPV. Similarly in the study of Wysocki,[15] improvement in P_{aO2} after 1 hour of NPPV did not predict subsequent successful outcome. Hence, early improvement in gas exchange does not necessarily predict a favorable outcome in the subsequent course of NPPV.

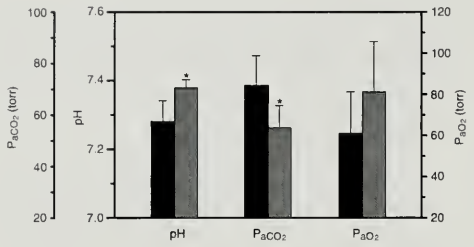

Fig. 4. Graph showing arterial pH, P_{aCO_2}, and P_{aO_2} before (black bar) and after (gray bar) receiving noninvasive positive pressure ventilation in successfully treated patients. * $p < 0.01$ (unpaired t-test) for comparison between before and after treatment. Data are from References 6-22.

In summary, in highly selected patients, NPPV appears effective in the treatment of acute ventilatory failure with an overall success rate of approximately 70%. The patient-ventilator interface (ie, nasal vs facial mask) or the underlying disease state has little influence on the success rate. Poor results may be expected in patients with hypoxemic respiratory failure. Pressure-preset ventilation may be more advantageous than VPV. The timing of applying and discontinuing NPPV, the duration, and early predictors for success remain to be established. More controlled trials are needed to assess the efficacy of NPPV in avoiding endotracheal intubation, in reducing intensive care unit (ICU) and hospital length of stay, in lessening mortality, and in reducing costs.

NPPV for Patients Who Refuse Intubation

Meduri and co-workers[23] recently reported the efficacy of NPPV in 11 patients with acute ventilatory failure who had refused intubation because of terminal illness (n = 4) or prior experience with intubation (n = 7). Of the 11 patients, 9 had hypercapnic ventilatory failure and 2 had hypoxemic respiratory failure. Successful outcome was defined as surviving ICU stay and correction of acidosis in the patients with hypercapnic respiratory failure or correction of hypoxemia with a reduction in F_{IO2} by ≥ 30% in the patients with hypoxemic respiratory failure. Mean duration of NPPV was 44 h (range, 11-88 h). Seven patients (64%) were discharged from the ICU (successful outcome), but only 5 of the 11 patients were discharged home, for a survival rate of 45%. Benhamou and co-workers[14] also studied a subgroup of patients (n = 17) in whom endotracheal intubation was contraindicated because of age and the family's request. In their study, 10 of 17 patients (59%) were successfully treated with NPPV, but only 47% of the patients survived. In both studies,[14,23] the immediate hospital survival rate remained poor, reflecting the severe, irreversible underlying pulmonary disease. Both studies are uncontrolled, and conservative treatment might yield a similar survival rate. Under these circumstances, prolonging life unnecessarily raises ethical questions,[33] particularly when the application of NPPV is for the purpose of life-closure tasks.[24]

NPPV in Discontinuation from Mechanical Ventilation

To our knowledge, only two studies claim efficacy for NPPV in weaning patients from conventional mechanical ventilation. Both are uncontrolled studies.

In the first study, Udwadia and co-workers,[25] studied 22 patients (9 with restrictive thoracic or parenchymal disease, 6 with neuromuscular disease, 7 following cardiac surgery) who had failed multiple weaning attempts. Nine of the patients had a tracheostomy. The median duration on conventional mechanical ventilation was 31 days, with a wide range from 2 to 219 days. Criteria for instituting NPPV consisted of intact bulbar function with preserved cough reflex, minimal airway secretions, hemodynamic stability, a functioning gastrointestinal tract, low F_{IO2}, and ability to breathe spontaneously for 10-15 min. After meeting the above criteria, the patient was extubated and nasal NPPV (using volume-preset ventilator with supplemental O_2 of 1 to 2 L/min) was applied. In the patients with a tracheostomy, the cuff was deflated and the tube was occluded. If NPPV was well tolerated, decannulation was performed within 36 hours. NPPV was unsuccessful in 2 patients with pulmonary fibrosis, but it was unclear whether the patients required reintubation. The 20 patients who tolerated NPPV were discharged to the general medical ward within 48 h. Subsequently, 2 of those 20 patients required reintubation, for an extubation success rate of 82% (18/22). The remainder of the patients were discharged home, with a median hospital stay from the start of NPPV to discharge of 11 days (range, 8-13). Ten of the patients continued to use nocturnal NPPV in the home. The authors concluded that NPPV can be an effective weaning modality that is worth considering early in the course of management of patients with chest-wall disease, neuromuscular disease, and COPD. Given the low cost of the equipment and reduced need for ICU resources, NPPV may be cost-effective. Although the median duration on NPPV is approximately one third that of conventional mechanical ventilation, the time required for stabilization of the acute episode has to be taken into account. The patient population in the Udwadia et al study[25] was carefully selected and would most probably tolerate nasal NPPV and benefit from long-term therapy.[3,4] Standard weaning criteria prior to transfer to NPPV are not available. With NPPV, patient cooperation, by and large, determines its success rate. NPPV tolerance cannot be safely predicted prior to extubation. Premature extubation for the purpose of weaning with NPPV carries a high risk for reintubation (approximately 30%, based on the overall success rate of 70%

in the treatment of acute ventilatory failure) unless patients are carefully selected.

In the second study, Restrick and colleagues[26] reported 14 patients (8 with COPD, 4 with neuromuscular disease, 2 post surgical) who had or were predicted to have weaning difficulties with standard weaning techniques (ie, PSV, CPAP, synchronous intermittent mandatory ventilation [SIMV], or T-piece). The median duration of conventional mechanical ventilation was 14 days, again with a wide range between 1 and 229 days. Three (2 COPD and 1 kyphoscoliosis) of the patients predicted to have weaning difficulties were extubated after 24 hours and treated with NPPV. This raises the question of whether these patients should have had a trial of NPPV from the outset of the respiratory failure, thus avoiding intubation. Four patients either had already been extubated (3) or decannulated (1), and one patient with a tracheostomy had mechanical ventilation discontinued prior to the application of NPPV. NPPV was applied in three episodes of self-extubation. Two patients had breathed through a T-piece and were transferred immediately to NPPV following extubation. The remaining patient was extubated at a SIMV rate of 2 breaths/min. One patient had two trials of NPPV, resulting in 15 trials of NPPV in the 14 patients. Criteria for the institution of NPPV were SIMV rate of < 5 breaths/min, or pressure support of < 10 cm H_2O; adequate cough reflex and absence of sputum retention; optimal nutritional status; and resolution of the precipitating factor leading to the respiratory failure, including radiographic changes. Nasal NPPV was applied with a volume-preset ventilator (a pressure-preset ventilator was used with one patient). NPPV was initially applied continuously, except during meals, physiotherapy, or nebulized bronchodilator treatment. NPPV was successful in 13 of the 15 trials for a success rate of 87.5%. In the patients who failed, one required reintubation and one had a tracheostomy through which portable mechanical ventilation was applied. In this study the majority of the patients (all but one) had been extubated, and 2 patients tolerated T-piece, the duration of which was unknown. In essence, NPPV was utilized to avoid reintubation (extubation failure) with a relatively high success rate. Both Meduri and co-workers[9] and Wysocki and co-workers[15] found a similar success rate in their patients who received NPPV because of post-extubation failure. NPPV was successful in 6 of 7 patients (86%) in the study of Meduri et al[9], and in 5 of 6 patients (83%) in the study of Wysocki et al.[15]

Considerations for Use of NPPV in Facilitating Weaning from Mechanical Ventilation

- A controlled study comparing the efficacy of NPPV with standard weaning technique (ie, SIMV, PSV, CPAP, T-piece) on weaning outcome, ICU and hospital length of stay, mortality, and costs is not available. Until such a study is available and the efficacy of NPPV to facilitate weaning is proven, attempted weaning from conventional mechanical ventilation) using NPPV when extubation failure is anticipated is deemed experimental and is not a standard practice.
- NPPV should not be used to facilitate weaning unless the patient meets the criteria listed in Table 2, and unless the precipitating factor leading to the ventilatory failure has resolved, and the patient has optimal nutritional status and is able to breathe spontaneously without any assisted device for a period of time (10-15 min).
- NPPV appears to be useful for avoiding intubation when acute ventilatory failure develops in the immediate post-extubation period. Under this circumstance, NPPV is worth trying, if one recognizes the lack of controlled trials.

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Long-Term Mechanical Ventilation and Weaning

David J Pierson MD

I. Introduction
II. Prolonged Mechanical Ventilation in the ICU
III. Current Options for Long-Term Management of the Ventilator-Dependent Patient
IV. Weaning after Prolonged Mechanical Ventilation in Acute Respiratory Failure
V. Weaning in Long-Term Mechanical Ventilation
VI. The Unweanable Patient
VII. Weaning vs Extubation
VIII. Conclusions

Introduction

In the intensive care unit (ICU), the term weaning is commonly used to denote the process of discontinuing mechanical ventilation regardless of the clinical setting or speed at which this is carried out.[1-5] This is at variance with the concept of weaning as it originates in mammalian biology, and many authorities believe the term should only be used to refer to the gradual withdrawal from ventilatory support in patients who cannot be taken off quickly.[6]

Long-term mechanical ventilation (LTMV) as encountered in home care refers to a means of support in patients who are not acutely ill and for whom such ventilatory support is presumed to be permanent.[7-10] In this context, weaning may not seem an appropriate topic for consideration. However, the term weaning may be used to refer not only to removal from ventilatory support but also to reduction (if not discontinuation) in support and/or invasiveness. In this context, clinicians involved in LTMV need to know what weaning involves, what the physiologic and other factors are that determine the need for ventilatory support, and potential reasons for unweanability in the process of providing the appropriate amount of support for the patient.[11,12]

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In this review, I address the gap between the ICU and acute illness, and the home and long-term stability. Occupying this gap in today's health care system are facilities with a variety of names whose purposes are to care more efficiently for ventilator-dependent patients who are not yet fully recovered from acute illness and to achieve maximal independence from ventilatory assistance for such patients as they stabilize.[9,13-16] Once such stabilization has occurred, weaning may be considered as *determining the medically appropriate level of ventilatory support*, that is, establishing a level that permits the greatest degree of ventilator-independence while also preventing physiologic deterioration.

Prolonged Mechanical Ventilation in the ICU

For perhaps 90-95% of all patients who receive mechanical ventilation in the ICU, the need for ventilatory support is short-lived, and the latter can be discontinued within a few hours to 2-3 days. This is true for individuals with normal underlying pulmonary function, and, perhaps to a lesser extent, for patients with pre-existing chronic lung disease (Fig. 1). Exact figures cannot be cited because they would differ among institutions and between different ICUs within a given institution, according to the differing patient populations served.[5]

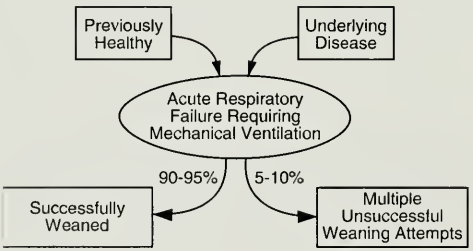

Fig. 1. Weaning outcomes for patients who survive an episode of acute respiratory failure requiring mechanical ventilation.

Weaning for this majority of patients who require only short-term mechanical ventilation is usually a straightforward process focused on respiratory factors such as gas exchange and pulmonary mechanics. Once the acute process requiring ventilatory support has resolved or substantially improved, the ability to resume spontaneous ventilation can be predicted with reasonable accuracy[17-19] or demonstrated empirically by progressive withdrawal of ventilatory support.[20-23]

However, for an occasional patient who would be expected to be able to resume spontaneous ventilation without difficulty and more frequently for those patients with severe underlying medical problems that place them in a category of expected weaning difficulties, one or several weaning attempts are unsuccessful because of hypoxemia, respiratory acidosis, increasing respiratory distress, or other signs of inability to breathe without assistance.[11,12] I believe that it is in this minority of patients that the art and science of weaning meet their greatest challenge.

Figure 2 places a given patient's prospects for successful weaning along a hypothetical continuum, from the virtual certainty of eventual liberation from the ventilator (100% likelihood of weaning—Point A in the figure), to inescapable dependence on mechanical support (0% likelihood of weaning—Point B in the figure). For some patients, such as those with high spinal cord injuries, completely spontaneous ventilation will clearly not be possible. On the other hand, a previously healthy patient recovering from acute respiratory distress syndrome (ARDS) who initially fails weaning attempts, in the face of continued high minute ventilation requirement and low respiratory system compliance, can most likely be assured of eventual success in weaning as the acute pulmonary process continues to resolve.

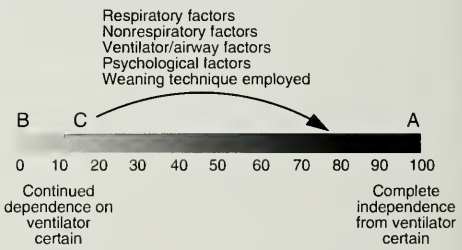

Fig. 2. Prospects for weaning after acute respiratory failure: A continuum from certain eventual liberation from the ventilator (Point A) to permanent ventilator dependency (Point B). Most patients fall somewhere between the two extremes (eg, Point C), and the clinician's task is to increase a given patient's likelihood of eventual ventilator independence through application of the factors shown above the curved arrow.

It is for the patients in the middle of the continuum in Figure 2 that weaning is the real challenge. For them, numerous factors in addition to gas exchange and pulmonary mechanics must be taken into consideration, including nonrespiratory medical factors, the properties and function of the ventilator and other apparatus being used, psycho-

logical and personality factors, and also the weaning technique employed and the skill with which it is pursued.

Even when initial attempts at weaning fail, the majority of patients will be able to be weaned and extubated within several days once their medical condition stabilizes and the factors just listed have been addressed. However, even in the most experienced hands a few patients remain who must be classified as unweanable, at least in the ICU setting. Some fail to improve, but yet survive for weeks with multiple organ dysfunction—thanks to the heroic efforts of their caregivers. Many of these chronically critically ill patients eventually die while still receiving mechanical ventilation and other forms of life support (Fig. 3). Others, however, reach a condition of medical stability despite being unable to be weaned, and for these individuals a variety of medical, psychosocial, and economic factors make it desirable that they be moved out of the ICU environment.[24]

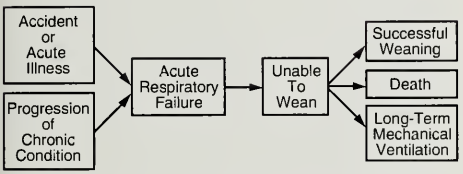

Fig. 3. Possible outcomes for the patient requiring prolonged ventilatory support for acute respiratory failure. Not every patient is a candidate for long-term ventilation: some remain chronically critically ill and unable to recover sufficiently for either weaning or long-term ventilatory assistance to be a reasonable alternative.

Current Options for Long-Term Management of the Ventilator-Dependent Patient

Several options exist for unweanable patients who are medically able to leave the ICU (Fig. 4) Some can return home, although this requires that they be clinically stable and have access to considerable medical, technical, financial, social, and family resources.[7,8,25-27] Others, whose prospects for functional recovery and quality of life are more limited, may be able to be transferred to a nursing home or other extended care facility that will accept mechanically ventilated patients. For the remainder, including those patients whose condition remains acute and who require close observation and nursing care, several types of facility exist, albeit with much regional variation in type and availability. These facilities differ in certain respects and are known by a variety of names (Fig. 4), including chronic ventilator units within acute care hospitals, long-term acute care facilities, noninvasive ventilator units, and regional weaning centers (RWCs).

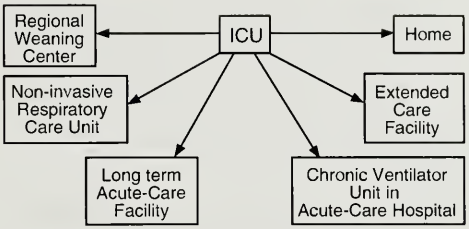

Fig. 4. Possible site alternatives to the intensive care unit (ICU) for patients requiring prolonged ventilatory support after an episode of acute respiratory failure.

In the RWC category, Scheinhorn and colleagues at Barlow Hospital in Southern California have recently reported the experience of their RWC with 421 consecutive patients transferred to them from acute care hospitals.[15] Two hundred forty-eight (59%) of these patients were women. They were predominately elderly, with a mean age of about 70 years. Eighteen patients who were transferred to other acute care facilities were excluded from analysis. Of the remaining 403 patients, 287 survived to be discharged from the RWC and 116 died. Complete independence from ventilatory support was eventually achieved in 212 of the 287 surviving patients (74%). The remaining 75 patients continued to require at least part-time ventilatory assistance.

The patients in this series had been ventilated, on average, about 50 days at the referring hospital prior to transfer to the RWC. Those who were successfully weaned were ventilated for 39 ± 2.6 days at the RWC, as compared with 102 ± 9.1 days in those who could not be weaned. Of the 212 successfully weaned patients, 102 went home and 110 were discharged to extended care facilities. Survival at 1 year was 20% (42 patients) in the former group and 11% (23 patients) in the latter.[15]

A major impetus for transferring ventilator-dependent patients from acute-care hospitals to facilities such as a RWC is to reduce the cost of caring for them.[24,26,28] Mean daily costs for ventilator-dependent patients in ICUs of acute-care hospitals have been estimated to be on the order of $3200,[14] as compared to $980 for the patients in the Barlow series while they remained in the RWC and on the ventilator.[15] Once the survivors were discharged from the RWC, calculated placement costs were $600 for those who were ventilator-dependent and living in extended care facilities, and $405 for those who were ventilator-dependent and living at home.[15] For the patients who were successfully weaned at the RWC, calculated daily costs after discharge were $275 for those at extended care facilities and $28 for those living at home.

Although the patients reported by Scheinhorn and associates[15] were highly selected and likely not representative of many individuals deemed unweanable in the acute-care setting, the generalizations can be drawn that the care of such patients costs less in facilities such as the RWC, and that a substantial proportion of patients can eventually be weaned from ventilatory support.

Weaning after Prolonged Mechanical Ventilation in Acute Respiratory Failure

Regardless of where the weaning attempt occurs in a patient who has required prolonged mechanical ventilation for acute respiratory failure, attention to a number of important principles increases the likelihood of ultimate success, thus effectively moving the patient represented by C in Figure 2 farther to the right in the diagram. As mentioned previously, readiness for resumption of spontaneous ventilation can usually be assessed by examination of gas exchange and pulmonary bellows function. Once the patient has required ventilatory support for a period of weeks, however, attention to nonrespiratory factors assumes paramount importance.[29-31] These nonrespiratory factors in weaning (Table 1) emphasize the fact that patients with prolonged acute respiratory failure typically develop multisystem dysfunction and are prone to a variety of abnormalities that can interfere with weaning.

Table 1. Nonrespiratory Factors in Weaning

Acid-base status
Respiratory alkalosis: decreased ventilatory drive
Metabolic alkalosis:decreased ventilatory drive
Metabolic acidosis: increased ventilatory demand
Mineral and electrolyte balance
Hypophosphatemia: ventilatory muscle weakness
Hypomagnesemia: ventilatory muscle weakness
Hypokalemia: ventilatory muscle weakness
Hypothyroidism: decreased ventilatory drive; impaired muscle function
Medical stability in other organs and systems
Cardiac: excessive preload (ie, overall volume overload; increased preload on discontinuation of positive-pressure ventilation); impaired contractility
Renal: renal insufficiency; metabolic acidosis
Hepatic: encephalopathy; protein synthesis
Gastrointestinal: stress-related hemorrhage; ability to take enteral nutrition
Neurologic: level of consciousness; ability to protect airway and clear secretions
Effects of drugs: narcotics; benzodiazepines; other sedatives/hypnotics; muscle relaxants; aminoglycosides
Nutritional status
Ventilatory muscle function
Ventilatory drive
Immune defense system
Psychologic and motivational factors

In prolonged acute illness, weaning depends on the balance between ventilatory demand and the patient's capabilities. A common reason for unweanability is that the minute ventilation required to keep the patient's P_{aCO_2} in the normal range is too high (eg, 16-18 L/min instead of the expected 10 L/min). In such cases, one or both of two possible physiologic derangements must be present, assuming that the patient has a normal P_{aCO_2} and is not being hyperventilated: hypermetabolism (increased CO_2 production, $\dot{V}_{CO_2}$) or inefficient ventilation (increased dead space ventilation, V_D/V_T).

Causes of hypermetabolism include fever, sepsis, excessive skeletal muscle activity (as with shivering, seizures, or agitation), or overzealous caloric supplementation, especially with carbohydrate calories. Inefficient ventilation (increased V_D/V_T) is a hallmark of severe obstructive lung disease and is a characteristic finding late in the course of ARDS; in either condition it can also result from dynamic hyperinflation in association with excessive positive end-expiratory pressure (PEEP), either dialed-in or endogenous (auto-PEEP). Bedside measurement of both $\dot{V}_{CO_2}$ and V_D/V_T requires a 3-minute exhaled gas collection and a simultaneous arterial blood gas determination, and can be helpful to the clinician in identifying the cause or causes of excessive minute ventilation requirement.[1,31]

Drugs can interfere with weaning either by suppressing ventilatory drive or by compromising ventilatory muscle function. All sedatives, tranquilizers, and hypnotics depress ventilatory drive, albeit to varying degrees. Muscle relaxants such as pancuronium, vecuronium, and atracurium, often used as adjuncts to ventilatory management, can cause muscle weakness and prolong weaning in two ways: (1) clearance of these agents may be prolonged for days in the presence of renal insufficiency, and (2) a recently described myopathy, most often seen with concurrent systemic administration of corticosteroids, can produce muscle weakness that lasts for many weeks.[32] Aminoglycoside antibiotics can also produce neuromuscular paralysis, although this is rarely seen today.

Each of the other factors listed in Table 1 can delay or prevent weaning in prolonged acute respiratory failure. The clinician must be methodical in reviewing each possible cause for weaning failure when initial attempts at discontinuation of positive-pressure ventilation are unsuccessful.

Weaning in LTMV

For patients no longer in acute respiratory failure, in whom general medical stability has been achieved, the concept of weaning is different from those in the settings

already discussed. As shown in Table 2, the goals of weaning in LTMV are different than those in acute illness. Once medically stable and in a nurturing environment, the patient who is receiving ventilatory assistance can sometimes be weaned entirely or can have the amount of ventilatory support reduced in any of several aspects, even though complete weaning may not be possible. As shown by the extensive experience of Bach and associates,[33-36] such patients can often achieve considerable independence from mechanical devices while still receiving LTMV for at least part of each day.

Table 2. Goals for Weaning in Long-Term Mechanical Ventilation

Reduce amount of support
Decrease invasiveness of support
Increase independence from mechanical devices
Preserve function
Maintain medical stability

Because the goals of weaning in LTMV include the preservation of function and the maintenance of medical stability in addition to a reduction in mechanical support, complete discontinuation of all ventilatory assistance may not necessarily be a desirable goal for these patients. Weaning from LTMV altogether may lead to more rapid progression of the underlying chronic condition, with recurrence of acute respiratory failure. Thus, consideration of weaning in the patient requiring long-term ventilatory assistance must seek to determine that level of ventilatory support—be it total, partial, or none—most consistent with the patient's best overall medical interests.

Finding the right level and schedule of ventilatory assistance for a given patient is more art than science,[16] and replicating the notable results achieved in a few centers with experience managing hundreds of such patients[35,36] may not be possible for clinicians attempting to do this for the first time—or even the tenth. Two points that I believe are agreed upon by those with the most experience in this field are: first, that optimal management for a given patient must be individualized rather than attempted according to some fixed protocol, and, second, that multiple devices and techniques must be available. One patient may achieve increased independence from the ventilator through the use of a Pneumobelt,[37,38] while the next may be unable to tolerate this device but be quite successful once trained in glossopharyngeal respiration ("frog breathing").[39] A third patient, continuously ventilator-dependent, may be able to switch to totally noninvasive intermittent positive-pressure ventilation via mouthpiece or facemask, permitting closure of a long-present tracheostomy.[35,36]

Several excellent recent reviews provide more comprehensive examination of these and other possibilities for less-invasive long-term ventilatory assistance than is possible in this brief discussion.[36-38,40]

The Unweanable Patient

Discontinuation of mechanical ventilation may not be attainable for several reasons. Neuromuscular function may be inadequate to support the patient's ventilatory needs, as in high spinal cord injury and the late stages of muscular dystrophy (Point B in Figure 2). Other patients may never be brought to a medically stable state, remaining chronically critically ill despite all the efforts of caregivers to treat identified acute problems. Less well defined is another category of patients who may be unweanable because of impaired neuropsychologic function, which in some cases may be amenable to therapeutic intervention but in other instances may not.[25,41-43]

Weaning may be unsuccessful for reasons other than those pertaining to the patient per se. Although no properly controlled studies of different approaches to weaning exist, the clinician's approach to discontinuation of ventilatory support may be sufficiently inappropriate to the patient's physiologic state and psychosocial needs as to render unweanable a patient who with other clinical approaches might successfully have ventilation discontinued. Finally, as discussed elsewhere in this issue by Sassoon,[44] the use of inappropriate equipment, in the form of the ventilator and its associated apparatus, in attempting to wean patients from long-term mechanical ventilation, can effectively preclude success in this endeavor.[6,44-46]

Weaning vs Extubation

Weaning is the discontinuation of ventilator support, and extubation is the removal of an artificial airway from the patient's respiratory tract.[47] Despite these fundamental differences, the literature on ventilator weaning contains much confusion with respect to these two terms and these two processes.[5] Although weaning and extubation can frequently be carried out together (for example, following short-term ventilatory support for recovery from anesthesia or uncomplicated respiratory failure), settings exist in which it is crucial to distinguish between the two. In the acute setting, airway protection may be necessary after the need for ventilatory assistance has disappeared, as in acute epiglottitis or upper airway injury associated with smoke inhalation.

Intubation may be necessary occasionally to facilitate secretion removal in patients who can otherwise ventilate adequately and protect their upper airways.[48] In the setting of long-term mechanical ventilation, it may be possible in some circumstances to extubate or decannulate the airway, and yet continue ventilatory support, as has been shown

impressively by the work of Bach et al.[35,36] As indicated in Table 2, one goal of weaning in LTMV should be to decrease the invasiveness of such support, so long as doing so does not lead to clinical deterioration.

Conclusions

Weaning from mechanical ventilation is both conceptually and practically different in different clinical settings. In LTMV, weaning seeks to attain the least mechanical support that is consistent with maximal function and to accomplish this in the least invasive manner. Because patients requiring LTMV have varying degrees of impairment of airway protection, ventilatory muscle function, and other components of successful ventilation, total weaning may not be an appropriate goal for a particular patient. Clarification of a patient's ultimate weaning status and achievement of these goals generally will not be possible in the acute-care hospital setting.

The ultimate degree of independence from ventilatory support may not be predictable in the short term. Ultimate weaning will depend not only on the physiologic and technical considerations discussed in this article but also on less tangible aspects of the patient's illness and the care provided, including the patient's psychosocial status and the home environment. Weaning also depends greatly on the knowledge and skills of those managing the patient long term.

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Neonate with Right Hemithorax Opacification

Steven E Sittig RRT

A 38-week-gestation infant girl was born via normal spontaneous vaginal delivery without complications. The amniotic fluid was clear, and Apgar scores[1] were 6 at 1 minute and 7 at 5 minutes after delivery. The infant's heart rate and pattern, respiratory rate, and pulse oximetry oxyhemoglobin saturation (S_{pO_2}) were monitored continuously. The patient developed respiratory distress immediately after birth, evidenced by laryngeal braking (grunting), and was given supplemental oxygen—F_{DO_2} 0.6— by oxyhood. At one hour of age, her S_{pO_2} began decreasing. On physical exam, breath sounds were absent over the right lung, and her abdomen was unremarkable except that the liver margin was low; she was vigorous and crying. Capillary refill was < 3 seconds and mean arterial blood pressure was 45 mm Hg. A catheter was placed in an umbilical artery and an anteroposterior chest radiograph was taken (Fig. 1).

Initial umbilical-artery-blood analysis revealed P_{aO_2} 69 torr, P_{aCO_2} 72 torr, pH 7.08 and S_{aO_2} 90%. Continuous positive-airway pressure and F_{DO_2} 1.0 was begun at the community hospital and maintained until our transport team arrived at which time, the infant was intubated and intermittent mandatory ventilation via a transport ventilator was begun. The settings were peak inspiratory pressure 20 cm H_2O, positive end-expiratory pressure 3 cm H_2O, cycling rate 60 breaths/min, inspiratory time (t_I) 0.2 s, and F_{DO_2} 1.0.

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How would you answer these questions?

What abnormalities are present on the chest radiograph shown in Figure 1? _____

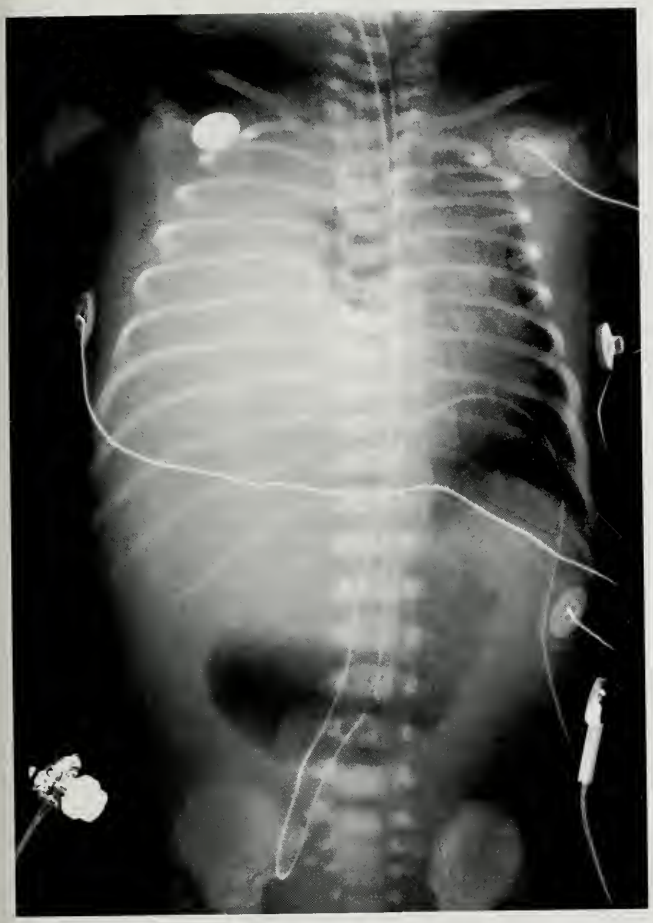

Fig. 1. Anteroposterior chest radiograph of a newborn infant girl with respiratory distress.

What diagnoses should be considered given the history, physical exam, and laboratory and radiographic evidence?

_____

_____

_____

_____

What other therapeutic and diagnostic procedures would you suggest? _____

_____

_____

_____

_____

Answers

Radiographic Findings: The right hemithorax is completely opaque, a pattern of bowel gas in the abdomen is not apparent, and the mediastinum has shifted to the left. The absent breath sounds noted on the physical exam are consistent with this appearance. In addition, the umbilical artery catheter is at L-2 (ie, the second lumbar vertebra) and the endotracheal tube tip can be seen at T-2 (ie, the second thoracic vertebra)—both are optimally placed. Transcutaneous attachment sites for a noninvasive blood-gas monitor and lead patches for the cardiorespiratory monitor can also be seen.

Differential Diagnosis: Based on available information, the differential diagnosis should include congenital diaphragmatic hernia (CDH), cystic adenomatoid malformation, or congenital tumor (ie, teratoma). Duodenal atresia may also be present .

Other Procedures: An ultrasound was performed and confirmed the presence of bowel and liver in the right hemithorax. This confirmed the diagnosis of right-side CDH. The patient was taken to the operating room where a 2 cm^2 diaphragmatic defect was repaired. It was noted that the small intestine and the lateral portion of the liver extended into the chest cavity. After surgery, the patient was sedated and paralyzed and ventilation was controlled. Her blood gases-pH were monitored frequently to detect pulmonary hypertension.

Discussion

In CDH, there is a displacement of the abdominal viscera through the diaphragmatic defect into the chest cavity. This defect is believed to occur by the sixth week of gestation and, therefore, because the bowel in the chest cavity occupies space that is required for the lung to develop normally, the lung is hypoplastic—less than fully developed— and the associated vasculature is affected.

The incidence of CDH is 1 in 2,000 live births and the defect is life-threatening, resulting in a 40-60% mortality.[2] The risk of mortality depends on several factors such as the degree of pulmonary hypoplasia, pulmonary hypertension, and age of onset of symptoms.[3-6]

The most common CDH is the Bochdalek hernia.[1,7,8] An unknown error in fetal development results in incomplete closure of the posterolateral portion of the diaphragm known as the pleuroperitoneal canal or foramen of Bochdalek. Of all CDHs, 85-90% are of this type with 80% on the left side, 15% on the right side, and 5% bilateral.[8,9] Right-side CDHs are of two types: the Bochdalek, just described, and the Morgagni. In the latter, the defect is found in the anterior peristernal area, is rarely diagnosed in newborns, and is not associated with cardiopulmonary decompensation. Often, a small portion of the liver is all that is displaced into the chest cavity.[9]

The Diagnosis

Infants with CDH commonly present with tachypnea, retractions, grunting, cyanosis, and cardiac dextroposition. The most severely affected newborns are symptomatic at delivery or within minutes of delivery. The symptoms become more severe as the infant's gastrointestinal tract fills with air, causing the herniated bowel and stomach to distend further, the mediastinum to shift toward the unaffected side, and severe respiratory distress to ensue. The physical exam reveals an enlarged anteroposterior chest diameter and a scaphoid or sunken abdomen. Although breath sounds are absent or diminished on the affected side, air movement in the unaffected lung can be transmitted to the affected lung and mislead the clinician. Bowel sounds may be heard over the thorax.

Suspected cases of CDH are usually diagnosed postnatally with radiographs of the chest and abdomen. A confirmatory film is likely show bowel gas in the chest on the affected side and an airless abdomen. However, the radiograph that is taken within minutes of delivery may show an opacified hemithorax because air has not yet entered the bowel. The mediastinum and the cardiac silhouette are shifted away from the affected side. The lung on the affected side is poorly aerated and air may only be visible at the apex and near the costophrenic angle on the unaffected side. If the tip of a nasogastric tube placed in the stomach can be seen in the chest (on radiograph) the diagnosis is confirmed.[10,11]

More often now, CDH is diagnosed prenatally by ultrasound. Sonography is accurate 90% of the time and has become part of routine obstetric care.[12] The absence of an intra-abdominal stomach bubble, polyhydramnios, or mediastinal shift detected by fetal ultrasound[13] should prompt a more thorough examination to look for evidence of abdominal organs in the fetal chest cavity.[14,15] It has been shown that fetal ultrasonography can detect CDH as early as 17 weeks gestation.[12]

The Repair

Until recent years, emergency surgical repair was the standard approach to infants with CDH. Because unstable infants undergoing immediate corrective surgery have a high mortality, new data suggest that a prolonged preoperative stabilization and delayed repair may be the key to successful management.[7,16-18] It has been suggested by Sakai et al[19] that the poorly compliant lungs are worsened by immediate surgical repair. In his study he measured lung

compliance in 9 patients who had undergone CDH repair. He found that after surgery, compliance decreased between 10 and 77%; four of the patients had > 50% reduction in compliance. In a study by Nakayama and colleagues,[15] pulmonary function tests were completed on 22 infants undergoing repair for CDH. Those whose defect was repaired immediately showed only slight improvement in compliance. In a follow-up study, Nakayama et al[20] found that airway compliance was twice the normal value in patients who had been stabilized for 1 week prior to repair. In those cases where surgical repair cannot be delayed due to the infant's unstable condition, some surgeons recommend preoperative extracorporeal membrane oxygenation (ECMO), either correcting the defect while on ECMO or after a successful course of ECMO.[21] However, one of the primary causes of morbidity and mortality in the postoperative management of these patients has been bleeding from the surgical site due to the use of heparin in the ECMO circuit. Major bleeding has been reported to occur in 50% of the patients and to contribute to death in 31% of cases.[22]

A successful outcome requires rapid preoperative assessment and preparation. When possible, the mother should be transferred before delivery to a medical center where pediatric surgeons, neonatologists, ECMO, and critical care specialists are available.[23] Maternal transport is safer than resuscitation and transport of a critically ill neonate.[24-26]

Respiratory Support

Immediately after delivery the initial resuscitation should begin with intubation. The use of bag and mask ventilation should be avoided as it tends to fill the bowel with air, worsening the bowel distention and further shifting the mediastinum. A nasogastric or orogastric tube should be placed and hooked to suction to provide intestinal decompression. The goals of ventilation are to prevent hypoxia, acidosis, and hypercarbia while decreasing the occurrence of pulmonary air leaks such as pneumothorax. The infant should be ventilated using the lowest mean airway pressure to ventilate and oxygenate.[27,28] Peak-airway pressures should not exceed 40 cm H_2O to decrease the risk of pulmonary air leaks from overinflation of the hypoplastic lungs. Other ventilator parameters should include 100% oxygen, rates of 80 to 150 breaths/min with short t_I (0.1-0.2 s).[13,28,29] The use of a high-frequency oscillatory ventilator may also be useful in adequately ventilating such infants.[30,31] Pharmacologic intervention may be required (sedatives and/or paralyzing agents) to facilitate ventilation.[2,4,27] It is also important to maintain a neutral thermal environment to minimize oxygen demand and to be diligent to detect pulmonary air leaks of any type.

Cardiovascular Support

Both preoperative and postoperative management should be directed toward avoiding or correcting pulmonary hypertension. It is not uncommon for infants with CDH to exhibit a period of excellent oxygenation either preoperatively or postoperatively—a 'honeymoon' period— that is quickly followed by a rapid decline in oxygenation owing to the onset of pulmonary hypertension.[32] With pulmonary hypertension, high resistance to blood flow in the pulmonary vascular bed causes right to left shunting of blood through the patent foramen ovale and ductus arteriosus. This pattern of blood flow is termed persistent fetal circulation (PFC) or primary pulmonary hypertension of the newborn (PPHN).[10] Once this pattern of shunting begins, it can be difficult to interrupt. The shunting associated with PPHN leads to acidosis, hypoxia, and cyanosis, which further stimulate pulmonary vasoconstriction and hypertension. Shunt is defined ≥ 20 torr difference between P_{aO_2}s from an artery supplied by preductal circulation (one emanating from the aorta between the aortic trunk and the ductus arteriosus) and a vessel supplied by a postductal artery (one emanating from the aorta between the ductus arteriosus and the descending limb of the aorta). The most important clinical challenge offered by patients with CDH is the avoidance or correction of pulmonary hypertension. The pharmacologic management of the PPHN associated with CDH includes systemic vasopressors, selective pulmonary vasodilators, and sedatives. Tolazoline is a selective pulmonary vasodilator that has been used widely to reduce pulmonary vascular resistance and shunting, but it never has been shown statistically to improve survival.[33,34] One of the main problems with tolazoline and with any other pulmonary vasodilator, is that they often affect systemic resistance as well. If a drug lowers the systemic pressure as much as it lowers the pulmonary pressure, the effective gradient across the ductus arteriosus remains the same, and the shunting from right to left is unaffected. The ideal drug would selectively dilate the pulmonary vasculature without affecting systemic blood pressure.

Recent work from a number of centers has shown that inhaled nitric oxide may reverse pulmonary hypertension.[35-37] Nitric oxide is a substance that has been measured in the lungs of mammals and relaxes the smooth muscle of the pulmonary vessels when administered endotracheally. Nitric oxide is transported bound to hemoglobin and breaks down so rapidly that it does not affect systemic blood pressure.[35]

As new techniques are shown to be effective, more infants are surviving CDH. Whereas the infant we report underwent almost immediate repair of the diaphragmatic defect, many clinicians believe that delayed repair—ie, until

the threat of PPHN is past—is preferred. Breaux et al[16] reported an increase in survival from 20% to 55% with delayed repair with or without ECMO. In that study, the average age at repair was 26.5 hours. In a 21-year follow-up study, Reid and Hutcherson[38] found that survivors led virtually unrestricted lives, with no limitation of activity due to lung function. Mild small-airways disease has been found in some of the infants.[38-39] In a study by Wohl et al,[39] survivors who were followed for 18 years had normal lung volumes and diffusion capacity. The total and 1-second forced vital capacity were 89% and 80% of predicted, respectively. ^{133}Xenon radiospirometry showed equal volumes of lung bilaterally, but blood flow to the hemithorax with the CDH was reduced in all patients.

It appears that vascular development never quite catches up with alveolar growth.[39] Falconer et al[40] reported a decrease in ipsilateral lung perfusion to 34% in patients who were ventilated more than 4 days. Peak expiratory flow and expired vital capacity were reduced by 50%.[40] Because these patients were not only ventilated but treated with ECMO, the relative contribution of the treatments to the morbidity is not clear.

The above information is important for us as respiratory care practitioners to understand when caring for a patient with a history of CDH. The infant whose case was presented here had a relatively uncomplicated postoperative course and was discharged directly home. Reportedly, the child is doing well as she approaches her second birthday.

ACKNOWLEDGMENT

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41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

Letters

Letters on topics of current interest or commenting on material in RESPIRATORY CARE will be considered for publication. The Editors may accept or decline a letter or edit without changing the author's views. The content of letters as published may simply reflect the author's opinion or interpretation of information—not standard practice or the Journal's recommendation. Authors of criticized material will have the opportunity to reply in print. No anonymous letters can be published. Type letter double-spaced, mark it "For Publication," and mail it to RESPIRATORY CARE Journal, 11030 Ables Lane, Dallas TX 75229-4593.

Not Turf Protection but Teamwork

> "Understandably they (respiratory therapists) worry that their narrow, specialized, and technical focus may make them obsolete in the future....I believe that nurses are the best prepared to offer the least expensive, most efficacious care possible....Therefore, other, more narrowly trained health care workers need not engage in cross-training to expand their scope of practice into nursing's..."[1]

As I read this statement in a nursing publication published in the Fall of 1994, I was left to wonder about the author's ability to work as a team member. Although I agree that health care reform necessitates the employment of the most efficient workers, I cannot support the author's assertion that the best equipped and most skilled health care workers can all be found in one particular field—be that nursing, respiratory care, cardiopulmonary care, physical therapy, or whatever. It is this type of narrow, protectionist thinking that will end in costing many hospitals their very existence. In order to remain competitive and viable, health care administrators need to look closely into all departments and select those individuals with the most to offer. It is those with the most to offer who can fill the future positions on the health care team, the majority of which have yet to be created. Their concern will not be to assign duties to representatives of one particular discipline, but rather to create teams of key players from many areas of expertise—players who have consistently demonstrated the ability to perform well. Those facilities that manage to put together the best, most efficient teams will be successful. Those with narrowly focused beliefs will find that they have made a costly mistake. TEAMWORK—that's the key!

Jack Marcelis RRT
Director of Respiratory Care
Vencor
Philadelphia, Pennsylvania

REFERENCE

1. Hopp L. RNS president's message. Perspectives in Respiratory Nursing 1994;Nov:2,11.

AARC

41st Annual Convention and Exhibition
December 2-5 • Orlando, Florida

1995 Call for Abstracts

RESPIRATORY CARE • OPEN FORUM

The American Association for Respiratory Care and its science journal, RESPIRATORY CARE, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present papers at the OPEN FORUM during the AARC Annual Meeting in Orlando, Florida, December 2-5, 1995. Accepted abstracts will be published in the November 1995 issue of RESPIRATORY CARE. Membership in the AARC is not necessary for participation.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) **an original study,** (2) **the evaluation of a method or device,** or (3) **a case or case series.** Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, health-care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract will be the only evidence by which the reviewers can decide whether the author should be invited to present a paper at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions.* Give specific information. Do not write such general statements as "Results will be presented" or "Significance will be discussed."

Essential Content Elements

An original study abstract *must* include (1) Introduction: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results. **A method/device evaluation** abstract *must* include (1) Introduction: identification of the method or device and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate. **A case report** abstract *must* report a case that is uncommon or of exceptional teaching/learning value and must include (1) patient data and case summary and (2) significance of case. Content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

Abstract Format and Typing Instructions

Accepted abstracts will be photographed. First line of abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institutions(s), and location. Underline presenter's name. Type or electronically print the abstract *single spaced in the space provided on the abstract blank.* Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. Make the abstract all one paragraph. Data may be submitted in table form *and simple figures may be included provided they fit within the space allotted.* No figures, illustrations, or tables are to be attached to the abstract form. Provide all author information requested at the bottom of abstract form. A clear photocopy of the abstract may be used. Standard abbreviations may be employed without explanation. A new or infrequently used abbreviation should be preceded by the spelled-out term the first time it is used. Any recurring phrase or expression my be abbreviated if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of RESPIRATORY CARE at (214) 243-2272.

Deadline Allowing Revision

Authors may choose to submit abstracts early. Abstracts received by March 15 will be reviewed and the authors notified by April 22. Rejected abstracts will be accompanied by a written critique that should in many cases enable authors to revise their abstracts and resubmit them by the final deadline (May 27).

Final Deadline

The mandatory Final Deadline is May 27 (postmark). Authors will be notified of acceptance or rejection *by letter only*—to be mailed by August 15.

Mailing Instructions

Mail (Do not fax!) 2 clear copies of the completed abstract form and a stamped, self-addressed postcard (for notice of receipt) to:

RESPIRATORY CARE OPEN FORUM
11030 Ables Lane
Dallas, TX 75229-4593

1995 Respiratory Care Open Forum
Abstract Form

18.8 cm or 7.4"

13.9 cm or 5.5"

1. Title must be in all upper case (capital) letters, authors' full names and text in upper and lower case.
2. Follow title with all authors' names including credentials (underline presenter's name), institution, and location.
3. Do not justify (ie, do leave 'ragged' right margin).
4. **Do not use type size less than 10 points.** (See reduction samples below.)
5. All text, tables, and figures must fit into the rectangle shown.
6. Submit 2 clean copies. This form may be photocopied if multiple abstracts are to be submitted.

Mail original & 1 photocopy (along with postage-paid postcard) to

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Early deadline is March 15, 1995 (abstract received)

Final deadline is May 27, 1995 (abstract postmarked)

Presenter's Name & Credentials

Presenter's Mailing Address

Presenter's Voice Phone & Fax

Corresponding Author's Name & Credentials

Corresponding Author's Mailing Address

Corresponding Author's Voice Phone & Fax

This is 14-point type, with necessary reduction.

This is 12-point type, with necessary reduction.

This is 10-point type, with necessary reduction.

This is 9-point type, with necessary reduction (too small!)

AARC SUMMER FORUM

Vail, Colorado July 14-16, 1995

RESPIRATORY CARE WEEK

October 1-7, 1995

AARC ANNUAL CONVENTION SITES & DATES

1995—Orlando, Florida, December 2-5
1996—San Diego, California, November 2-5
1997—New Orleans, Louisiana, December 6-9
1998—Atlanta, Georgia, November 7-10

OPEN FORUM 1995

It's time to submit your abstracts for possible presentation at the OPEN FORUM during the AARC Annual Meeting in Orlando, Florida, December 2-5, 1995.

An early deadline (March 15) allows opportunity for revision following review, with re-submission by the final deadline (May 27). For more information on changes to the rules/instructions, see Page 303 of this issue.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1995 Examination and Fee Schedule

	Examination Date	Application Deadline		Examination Date	Application Deadline
CRTT:	March 11, 1995	January 1, 1995	**CPFT:**	June 3, 1995	April 1, 1995
	July 15, 1995	May 1, 1995			
	November 11, 1995	September 1, 1995	**RPFT:**	December 2, 1995	September 1, 1995
RRT:	June 3, 1995	February 1, 1995	**Peri/Ped:**	March 11, 1995	November 1, 1994
	December 2, 1995	August 1, 1995			

Fee Schedule

CRTT	
—new applicant:	$ 90.00
—reapplicant:	$ 60.00
RRT Written and Clinical Simulation	
— new applicant:	$190.00
—reapplicant:	$160.00
Written Registry Only	
—new applicant:	$ 90.00
—reapplicant:	$ 60.00
Clinical Simulation Only—new & reapplicant:	$100.00
CPFT	
—new applicant:	$100.00
—reapplicant:	$ 80.00
RPFT	
—new applicant:	$150.00
—reapplicant:	$130.00
Perinatal/Pediatric Specialty	
—new applicant:	$150.00
—reapplicant:	$130.00

	Active	Inactive
CRTT Recredentialing:	$25.00	$ 60.00
RRT Recredentialing:		
Written Registry Examination	$25.00	$ 60.00
Clinical Simulation Examination	$65.00	$100.00
CPFT Recredentialing:	$25.00	$ 80.00
RPFT Recredentialing:	$25.00	$130.00
P/P Specialty Credentialing:	$25.00	$130.00
Membership Renewal:		
CRTT/RRT/CPFT/RPFT		$ 12.00
Credential Verification	$ 2.00	$ 15.00
Replacement Certificate	$ 6.00	$ 25.00
Copy of NBRC Directory: 1994		
Active Credentialed Practitioners	N/C	$ 25.00
Copy of NBRC 1994 Listing of All		
Credentialed Practitioners	$10.00	$ 25.00

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Calendar of Events

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month in which you wish the ad to run. Submit copy and insertion orders to Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane, Dallas TX 75229-4593.

AARC & AFFILIATES

March 12–15 in Denver, Colorado. The AARC and the National Jewish Center for Immunology and Respiratory Medicine announce the 5th International Conference on Pulmonary Rehabilitation and Home Ventilation at the Hyatt Regency Hotel. Pre- and post-conference postgraduate workshop topics include pulmonary rehabilitation, home ventilator care, respiratory sleep disorders, nicotine dependency treatment, and transtracheal oxygen. The program is endorsed by the American College of Chest Physicians; program chairman is Barry Make, MD. Contact the National Jewish Office of Professional Education at (303) 398-1000.

March 30–31 in Los Angeles, California. The CSRC Chapter 4, Queen of Angels–Hollywood Presbyterian Medical Center, and the Children's Hospital of Los Angeles present the 10th Annual Symposium, "Clinical Advances in Ventilator Technology." According to CSRC Chapter 4, this symposium meets continuing education requirements for all California RCPs. Contact Carrie Comorre, QAHP Medical Center, 1300 N Vermont Ave, Suite 810, Los Angeles CA 90027, (213) 664-6180

April 10–12 in Philadelphia, Pennsylvania. The Southeast District of the PSRC presents its 30th Annual Conference and Exhibition, "Meeting the Challenge," at the Adam's Mark Hotel. Topics covered in the 3-day seminar include issues regarding protocols, respiratory care in alternative work sites, specialized diagnostics, and new ventilator strategies. Contact Kathy Fallon at (215) 842-6590 or Denise McCardle at (215) 961-2131.

April 12–14 in Overland Park, Kansas. The Kansas Respiratory Care Society announces the 18th Annual Educational Seminar at the Overland Park Marriott. The seminar, entitled "Ch... Ch... Ch... Changes in Respiratory Care — Now and in the Future," looks at health-care reform as it relates to the respiratory care profession. The program is approved for 10 Category 1 CRCE credit hours. Contact Pat Munzer, 351 Woodbury Ln, Topeka KS 66606, (800) 332-0291, ext 1619.

April 17–19 in Bellevue, Washington. The Respiratory Care Society of Washington announces the 22nd Annual Pacific Northwest Regional Respiratory Care Conference at the Bellevue Red Lion Inn. Management, pediatrics, and education are featured subjects. The latest in health care equipment will be on display for two days. For a brochure, contact Bob Bonner, Chair, Health, Education, and PE Division, Highline Community College, PO Box 98000, Des Moines WA 98198, (206) 878-3710, ext 469.

April 25–28 in Cincinnati, Ohio. Region II for Respiratory Care (the Ohio, Kentucky, and Indiana societies) hosts its 22nd Annual Meeting, "Charting New Waters," at the Albert B Sabin Convention Center and Hyatt Regency Hotel. Contact Jeff Jones RRT, 1995 Chairman of Region II, at (513) 438-0388.

May 10–12 in South Padre Island, Texas. The Rio Grande Valley District of the TSRC presents its annual seminar at the Sheraton Beach Resort. Featured topics include neonatal and adult critical care and methods to enhance development of critical pathways. CRCE credit has been requested. The registration fee, which includes a banquet ticket, is $50; the cost of the scheduled fishing trip is extra. Contact TSRC, PO Box 2048, Harlingen TX 78551. The deadline for discount room confirmation at the Sheraton is April 9; call the resort at (800) 672-4747, and mention the seminar to receive a discount rate.

May 17–19 in El Paso, Texas. The Southwest Region of the TSRC presents its 24th Annual Seminar at the El Paso Airport Hilton. The theme is "Thriving in an Era of Change"; CRCE credit has been requested. Contact Carmen Castillo at Sierra Medical Center (915) 747-2771.

OTHER MEETINGS

March 21–22 in Durham, North Carolina. The Respiratory Care Departments of Duke University Medical Center, Durham Regional Hospital, Durham Technical Community College, and University of North Carolina Hospitals announce the 9th Annual Cardiopulmonary Technology Update at the Omni Durham Hotel & Convention Center. Dr Forrest Bird is the keynote speaker of the seminar, which features an exhibit hall displaying the latest advances in medical scientific equipment and services. Registration for the 2-day seminar is $70. Contact Thilo Hanisch RRT, or Kathy Waters RRT, at (919) 966-1336 or Chuck Alford at (919) 470-5366 or fax (919) 966-7647.

March 22–23 in Baltimore, Maryland. The Johns Hopkins Hospital Department of Respiratory Care presents a 2-day respiratory and critical care medicine seminar in the Turner Auditorium of The Johns Hopkins University School of Medicine. Topics include: ventilatory management of head and chest trauma, rehabilitation of ventilator-dependent patients, noninvasive mechanical ventilation, legal issues for the health care professional, invasive vs noninvasive monitoring in mechanical ventilation, hyperbaric oxygen therapy, high-frequency oscillation, treatment of chronic lung disease in children, surfactant therapy, pediatric ECMO, ribavirin uses and updates, synchronous ventilation in infants, and a cystic fibrosis update. Approval for 14 Category 1 hours of CRCE credit is pending. Registration for the 2-day seminar is $100 for AARC members and $120 for nonmembers. Contact Tony Bilenki at (410) 955-9276.

March 23–25 in Cleveland, Ohio. The Cleveland Clinic Foundation presents a continuing education program entitled

"Pulmonary & Critical Care Medicine" at The Cleveland Clinic's Bunts Auditorium. The course has been approved for Category 1 accreditation. Call The Cleveland Clinic Foundation at (800) 762-8173.

March 24 in Wilmington, Delaware. The department of respiratory care at the Alfred I duPont Institute presents "Tools for Pediatric Ventilation: Where We Are and Where We're Going...," a course about ventilation techniques and the management and identification of patients as candidates for various modes of ventilation. The course is approved for 6 Category 1 CRCE credit hours and up to 6 Category 1 credit hours of the Physician's Recognition Award of the American Medical Association. Contact the Alfred 1 duPont Institute Medical Education Department at (302) 651-6750.

April 5–7 in San Francisco, California. The California Society for Pulmonary Rehabilitation (CSPR) announces its annual conference at the Sir Francis Drake Hotel. A preconference workshop is offered April 5 from 1:30 p.m. to 5 p.m. for $30; participants may choose one of two workshop topics: transtracheal oxygen or new pulmonary rehabilitation. The April 6-7 conference fee, which includes two lunches, is $175 for CSPR members and $250 for nonmembers; topics include therapeutic strategies in asthma, advanced applications for transtracheal oxygen, reimbursement, and cardiac complications with pulmonary patients. Contact Gerilynn Connors BS RRT or Kathleen Morris MSN RRT at (707) 963-6588.

April 27–28 in Las Vegas, Nevada. The American Lung Association (ALA) of Nevada hosts the 10th Annual Respiratory Health Conference at the Tropicana Resort & Casino. Contact the ALA of Nevada at (702) 454-2500.

May 2–3 in Little Rock, Arkansas. Arkansas Children's Hospital presents the Diamond Conference on the hospital campus. Focus sessions on redefining physiotherapy and redirecting ventilatory strategies feature presentations and panel discussions. Roundtable lunch discussions are also planned. Abstracts will be accepted for poster presentation through March 1; selected abstracts will be chosen for slide presentation. Contact Mike Anders RRT at (501) 320-3535 or fax (501) 320-3411.

May 5–6 in Orlando, Florida. The Alpha$_1$ National Association (alpha$_1$-antitrypsin deficiency) hosts its 1995 National Conference at the Radisson Plaza Hotel. Featured topics include surgical choices for alpha$_1$ patients, new products, and alpha$_1$-related diseases. Contact Alpha$_1$ National Association, 1829 Portland Ave, Minneapolis MN 55404, (612) 871-1747.

May 20–24 in Seattle, Washington. The American Thoracic Society hosts its annual International Conference, featuring a variety of symposia and workshops on the prevention and control of lung disease. Contact 1995 International Conference, American Thoracic Society, 1740 Broadway, New York NY 10019-4374, (212) 315-8700.

June 16–19 in Toronto, Ontario, Canada. The Canadian Society of Respiratory Therapists (CSRT) announces its 30th Educational Forum at the Royal York Hotel.Workshop topics cover management, ventilation, cardiopulmonary diagnostics, lung transplantation, and research. Call (416) 368-2511 or fax (416) 368-2884 for more information.

October 29–November 2 in New York, New York. The 61st Annual International Scientific Assembly sponsored by the American College of Chest Physicians (ACCP) at the New York Hilton and Towers is entitled "Chest 1995: Prevention and Diagnosis of Chest Disease." Contact the ACCP, 3300 Dundee Rd, Northbrook IL 60062-2348.

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Part II • Prevention and Management of Ventilator-Induced Lung Injury
April 25, 12:30 p.m. to 2 p.m. Eastern Time—David J. Pierson, MD with Moderator Richard D. Branson, RRT

Part III • The Multidisciplinary Team Approach and Respiratory Care
June 20, 12:30 p.m. to 2 p.m. Eastern Time—Kevin L. Shrake, MA, RRT, CHE with Sam P. Giordano, MBA, RRT

Part IV • Emergency Respiratory Care: The Respiratory Care Practitioner's Ro
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Part V • Noninvasive Mechanical Ventilation: Its Role in Acute and Chronic Ventilatory Failure
August 2, 12:30 p.m. to 2: p.m. Eastern Time—Nicholas S. Hill, MD with Richard D. Branson, RRT

Part VI • Organizing a Respiratory Care Department without Walls
September 19, 12:30 p.m. to 2 p.m. Eastern Time—John R. Walton, MBA, MHA, RRT, CHE with Sam P. Giordano, MBA, R

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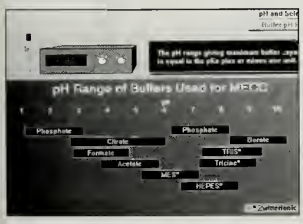
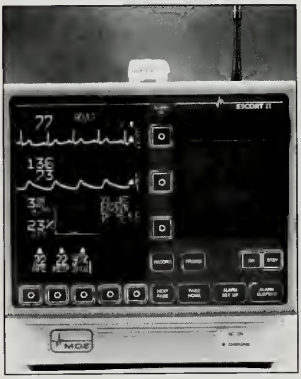
Passive-Gas-Exposure Monitor. AirScan Environmental Technologies Inc has launched a passive gas monitoring system that is fully integrated and compact; it provides on-site analysis without using an instrument or reader. According to AirScan, this easy-to-use format provides the user with outstanding convenience, accuracy, and precision in the palm of the hand. In addition to eliminating the time-consuming delays associated with laboratory analysis, AirScan monitors have a 24-month shelf life and require no refrigeration. The AirScan monitoring system, which exceeds OSHA requirements for accuracy and precision, is capable of measuring formaldehyde, glutaraldehyde, and ethylene oxide. Contact AirScan Environmental Technologies Inc, Dept RC, 197 Meister Avenue, Branchburg NJ 08876, (908) 725-1342. Don't forget to mention Respiratory Care when you call.



Computer-Based Training. SAVANT Audiovisuals Inc adds "Capillary Electrophoresis" to its computer-based training line. This 3-module Windows-based program includes *Principles of CE, Free Solution Capillary Electrophoresis Method Development, and Micellar Electrokinetic Capillary Chromatography Method Development.* According to the producer, these modules are interactive with maximum involvement and feedback. The modules run on IBM-PC compatible (386 or higher processor recommended) computers under Windows 3.0 or higher. Other training programs in laboratory techniques are also available from Savant Audiovisuals Inc, Dept RC, PO Box 3670, Fullerton CA 92634. Don't forget to mention Respiratory Care when you call (800) 472-8268.

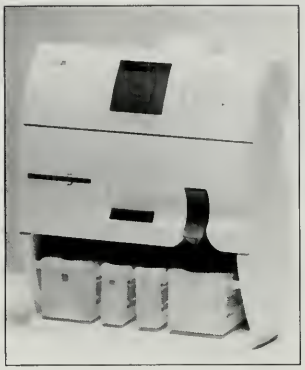
Ultrasound Medical Imaging. AXON' CABLE, a French cable manufacturer, announces a new series of ultra-miniature coaxial cables with diameters as small as 0.28 mm (0.011 in) for improved ultrasound medical imaging. The Pico-Coax series is designed for transducer-probe applications in medical imaging. Each cable connects to one of the transducer probe's sensors. The new cable design allows imaging equipment manufacturers to increase the number of sensors in each transducer probe, which in turn creates a clear image for more precise diagnostic information. Pico-Coax cables can transmit weak signals from the probes to the computer without distortion. According to the manufacturer, they are ideal for intracorporeal probes because of their small outer diameters. As many as 320 cables can be layered into a single, highly flexible bundle that is jacketed with either PVC, silicone, or polyurethane. Contact French Technology Press Office, Dept RC, 401 N Michigan Avenue, Suite 1760, Chicago IL 60611, or call (312) 222-1235. Please mention Respiratory Care when you call.



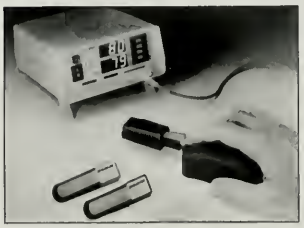
Hemodynamic Monitor. Medical Data Electronics announces expanded monitoring capabilities for its ESCORT II Series Portable Patient monitors. According to the manufacturer the option provides cardiac output determinations by the thermodilution method using proven algorithms licensed from the Edwards Division of Baxter International. The monitor also performs right-heart ejection fraction, stroke volume, and end-diastolic and

end-systolic volume determinations. The monitor displays each thermodilution curve and stores it for review and editing. Its capabilities include 2-lead electrocardiogram, invasive and noninvasive blood pressure, pulse-oximetry oxyhemoglobin saturation, end-tidal CO_2, and temperature. Contact Carol Burdette, (818) 768-6411, ext 2107. Please mention RESPIRATORY CARE when you call.

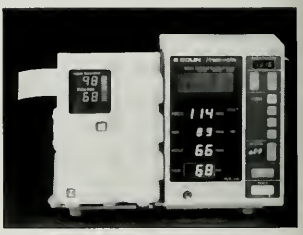
OXIMETER-PROBE COVER. BCI International introduces the OxiLink. According to the manufacturer, this disposable oximeter-probe-cover system maintains high standards of quality monitoring at a low-cost. The probe cover is a bright blue color to contrast against the skin for easy visibility and the physical design mimics the tapered contour of the finger. It has a microfoam lining that provides softness for patient comfort and is form-fitting to prevent dislodging. The OxiLink offers a unique polyurethane film that functions both as a barrier between the probe and the patient and ensures adhesion. Contact BCI International, Dept RC, W238 N1650 Rockwood Drive, Waukesha WI 53188-1199 or call (800) 558-2345. Please mention RESPIRATORY CARE when you call.



BLOOD GAS ANALYSIS. Radiometer America Inc announces the ABL 5 blood gas-pH analyzer. The manufacturer claims 5 essential qualities—(1) The ABL 5 measures pH, P_{CO_2}, and P_{O_2} from just 85 μL blood and calculates S_{O_2}, HCO_3^-, t_{O_2}, $P_{A\text{-}aO_2}$, and others. (2) Analysis is straightforward: open the inlet, aspirate the sample, and enter data. Operator menus provide clear instructions. (3) The analyzer provides accurate measurements and monitors its own performance. (4) Maintenance is easy and fast yet still meets stringent safety standards. (5) The ABL 5 is reliable, dependable, and durable. Contact Radiometer America Inc, Dept RC, 810 Sharon Drive, Westlake OH 44145, or call (800) 736-0600. Don't forget to mention RESPIRATORY CARE when you call.



OXIMETER TESTING SYSTEM. Nonin Medical Inc offers the Finger Phantom, an artificial finger designed to ensure consistent operation of pulse oximeters and sensors within the clinical range of 80-90% S_{PO_2}. The Phantom provides a reference for calibration of pulse-oximeter systems to satisfy quality-assurance standards of performance. According to the manufacturer, this product is compatible with most commonly used finger sensors and is supplied with a durable carrying case. Contact Nonin Medical Inc, Dept RC, 2605 Fernbrook Lane N, Plymouth MN 55447-4755 or call (800) 356-8874. Please mention RESPIRATORY CARE when you call.



MONITOR ENHANCEMENT. Colin Medical Instruments Corp announces that their Press-Mate vital-signs monitor can be coupled with the Nellcor N-20P pulse oximeter with a built-in printer, offering clinicians the ability to assess blood pressure, heart rate, temperature, and blood oxygen saturation noninvasively, using a single monitor. According to the company, values can be measured on demand, or the unit can be programmed to automatically monitor and record all data, including temperature, either continuously or at selected intervals. High- and low-alarm functions are included, and memory storage capacity is 152 lines of data. Colin Medical claims that it is the only company in the world to offer auscultatory, oscillometric, and arterial tonometry methods for the noninvasive measurement of blood pressure. Contact Carl Steffen (800) 829-6427 (voice mail, 6237). Please mention RESPIRATORY CARE when you call.

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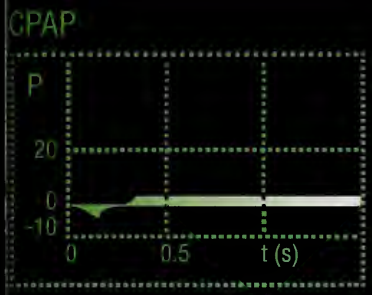

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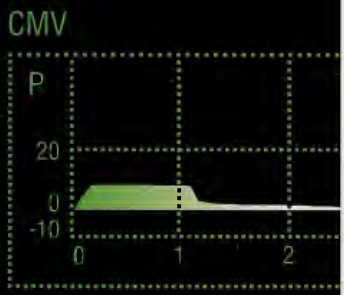

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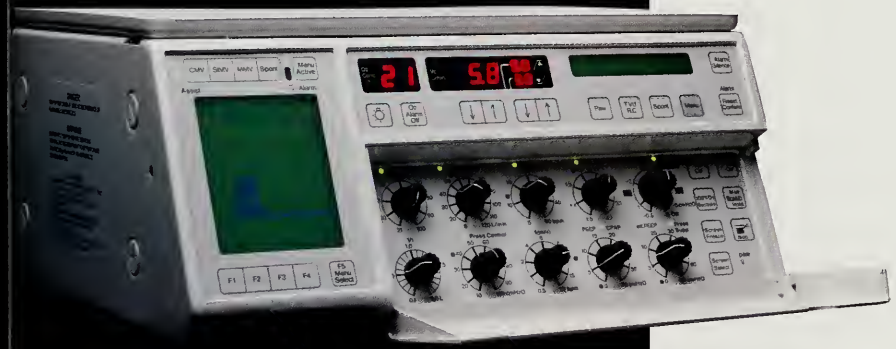

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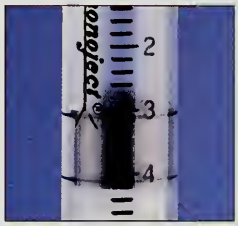

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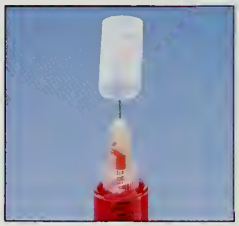

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